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Case Report

Who is the Mystery Man? Charles Bonnet Syndrome in the Setting of Age-Related Macular Degeneration

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Summary

Hallucinations are sensory experiences of something that is not actually present. They can occur in all five senses. Visual hallucinations are a clinical manifestation of neuro-ophthalmological dysfunction resulting from a wide variety of underlying etiologies. They can be very discomforting to some patients, regardless of their insight and can significantly decrease quality of life. Charles Bonnet Syndrome is an often-unrecognized condition where visual hallucinations occur in the setting of visual acuity or field loss. Clinicians can have the tendency to diagnose these patients with dementia or psychosis. Due to the lack of familiarity, it is possible that the syndrome is more prevalent than reported. This case concerns a 69-year-old male patient presenting with complex visual hallucinations, suggestive of Charles Bonnet Syndrome.

Background

Visual hallucinations are a clinical manifestation of neuro-ophthalmological dysfunction resulting from a wide variety of underlying etiologies. They can be very disconcerting to patients, regardless of their insight and can significantly decrease quality of life. The history, accompanying symptoms, and clinical signs are important elements for determining the most likely cause. In certain patients, further investigation may be necessary before a definitive cause can be determined. The Charles Bonnet Syndrome (CBS) refers to symptoms of visual hallucinations that occur in patients with visual acuity loss or visual field loss. Underlying conditions of vision loss associated with the CBS affect the eye, optic nerve, or brain and include a diverse set of pathologies, such as macular degeneration and stroke. The syndrome is not well recognized by clinicians and may often be misdiagnosed as psychosis or early dementia. We present a case of an elderly male with visual hallucinations and the pathophysiology and diagnosis of Charles Bonnet Syndrome.

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Case Presentation

69-year-old male with past medical history of coronary artery disease with three vessel coronary artery bypass done 15 years ago, hypertension, diabetes, hyperlipidemia, hypothyroidism, bilateral cataract surgery done six year ago came to the emergency room for evaluation of seeing an unknown person in his house for four days. Patient lived alone at home after his wife had passed one year ago. He described as seeing another man approximately his age, sitting in his living room across from him in his chair. The man was not familiar to him and would smile back to him, but not answer his questions. When he tried to close his eyes, the man would disappear. He would see the man in the evening and at nighttime, but not in the mornings. He would see the man intermittently for a few minutes and then he would disappear and reappear again. The man was seated in the same chair but was not always there.

He states that he called the police because of this issue and they searched the house finding no one. The next day he states that he had his sister come over to the house to see if she can see the man and she was unable to find him. The sister fearing that her brother was becoming psychotic called his primary physician who recommended he go the emergency room for evaluation of stroke. His wife had died a year ago, and he underwent normal grieving process and was not overtly depressed. He denied any cardiac symptoms, focal neurological deficits, episodes of confusion, convulsions fevers or neck rigidity. He and his sister denied any history of him having dementia. He denied any history of insomnia. He had been sleeping for 6-8 hours at night. He denied auditory and/or tactile hallucinations. He denied any history of psychiatric illness, drug or alcohol abuse. He was an ex-smoker who had quit 15 years ago after his bypass surgery. He did not have any clinical evidence of dementia. His medication list included aspirin, atorvastatin, lisinopril, metformin, levothyroxine and furosemide. He did not intake any form of benzodiazepine or antidepressants. He underwent an extensive neurological and psychiatric evaluation which was within the normal limits. There was no evidence of dementia or delirium. His eyesight was 20/60 in bilateral eyes with mild loss of central vision in bilateral eyes.

Investigations

He had extensive laboratory exam which revealed normal CBC, comprehensive metabolic panel, blood glucose, TSH, parathyroid hormone, vitamin B12, thiamine, folate levels. His urine drug screen was negative for recreational drugs and alcohol level was not

infarct. The echocardiogram showed normal left ventricular function and mild mitral regurgitation. Carotid duplex showed right internal carotid artery having 50-69% stenosis and left internal carotid artery having less than 50% stenosis with antegrade vertebral artery flow. MRI brain showed moderate diffuse atrophy with moderate chronic ischemic small vessel disease of the cerebral white matter. Small chronic right frontal cortical infarct.

An electroencephalogram was negative for epileptiform activity. Dilated eye examination using a slit-lamp instrument revealed round patches of depigmentation in the retina which revealed age related dry macular degeneration.

Differential Diagnosis

Patient did not have evidence of dementia, history of alcohol and illegal drugs consumption, brain tumors, seizures, delerium, acute stress, meningitis, Parkisnons disease, psychiatric illness, migraines and sleep disorders like insomnia or narcolepsy. Hence a diagnosis of Charles Bonnet Syndrome was entertained, given the ocular finding of age related dry macular degeneration. The patient was informed that the mystery man that he was seeing in his house was due to visual hallucinations from decreasing visual acuity.

Treatment

Patient was given a daily oral eye vitamin supplement to prevent progression of the macular degeneration. The patient was reassured that he was not having psychosis or dementia and that made him very relived. Hallucination suppressing techniques like rapid eye movements from one target to another, especially away from the affected visual field and blinking were taught. Increasing the illumination in the house was also recommended.

Outcome and Follow-Up

At six months follow up patient complains of few instances of visual hallucinations and he mentions that hallucination suppressing mechanisms that he was taught made himself-aware and he feels more in control of the situation.

Discussion

Visual hallucinations are a clinical manifestation of neuro-ophthalmologic dysfunction resulting from a wide variety of underlying etiologies. They can be very discomforting to some patients, regardless of their insight, and can significantly decrease quality of life [1]. A visual hallucination is a perception of an external visual stimulus where none exists. By contrast, a visual illusion is a distortion or modification of real external visual stimuli [2]. Visual illusions include distortions of size (micropsia or macropsia), shape (metamorphopsia) and color (dyschromatopsia).

Hallucinations can be classified as simple or complex which can narrow the differential diagnosis for the underlying cause [3]. Simple hallucinations are also referred to as “elementary” or “non-formed”. They do not include complex imagery. Examples include shapes, colors, lines, lights, or geometric designs. Simple hallucinations of light can be further classified as phosphenes, which are visual hallucinations of lights without structure, and photopsia, which are visual hallucinations of lights with geometric-like structure (triangles, diamonds and squares). Complex hallucinations can include images of people, animals, objects, or a lifelike scene. Complex hallucinations are also referred to as “formed”. Another classification scheme divides visual hallucinations into irritative phenomena that result in brief stereotyped hallucinations, and release hallucinations that are continuous and variable [4].

The Charles Bonnet Syndrome (CBS) refers to symptoms of visual hallucinations that occur in patients with visual acuity loss or visual field loss. These are often called release hallucinations, reflecting the most widely accepted theory of their pathogenesis. Charles Bonnet (March 13, 1720 - May 20, 1793) was a Swiss naturalist and philosopher and the first person to describe the syndrome. Initially, he observed symptoms of the syndrome in his 87-year-old grandfather, who was nearly blind from cataracts, yet still “saw” men, women, birds, carriages, buildings, scaffolding and tapestries before his eyes. In 1760, Bonnet described his eponymous syndrome, in which he documented a range of complex visual hallucinations that occurred in seemingly psychologically intact people [4].

Underlying conditions of vision loss associated with the CBS affect the eye, optic nerve, or brain and include a diverse set of pathologies, such as macular degeneration and stroke. While often not functionally disabling, the hallucinations can be distressing to patients and negatively impact quality of life [1]. Published case reports suggest that the syndrome is not well recognized by clinicians and may often be misdiagnosed as psychosis or early dementia [5,6]. The CBS may be more common than is generally appreciated. Visual hallucinations are often unreported by patients because they fear that they represent psychiatric disease [7-9]. When this symptom is specifically solicited in older patients with impaired vision, 11 to 15 percent admit to having visual hallucinations [10]. Release hallucinations have been reported in all age groups, including children [11]. However, most patients with CBS are older adults, the mean age is between 70 and 85 years [12]. This probably reflects the mean age at which the most common underlying conditions causing vision loss are seen.

Other conditions that are inconsistently found to be risk factors for CBS include cognitive impairment, cerebrovascular disease, cortical atrophy on brain imaging and social deprivation [13]. Patients with visual acuity loss or visual field loss from any cause, affecting any part of the visual pathway from the eye to the visual cortex, can develop visual hallucinations. Common underlying conditions include age-related macular degeneration, glaucoma, diabetic retinopathy and cerebral infarction [14,15]. However, these hallucinations have been reported to occur in virtually every acquired disorder affecting the visual system [4]. CBS does not occur with congenital blindness.

Hallucinations occur when visual sensory differentiation leads to disinhibition of visual cortical regions, which then fire spontaneously [16]. The content of the hallucinations was associated with specific regional activation that correlated with the known specialized function of that area of the visual cortex [17]. For patients with chronic ocular disease, the diagnosis of ocular disease is generally established for at least one year before hallucinations emerge [4]. The likelihood of release hallucinations increases with lower visual acuity. While there is no clear threshold of visual acuity loss, the prevalence of hallucinations appears to increase with acuity worse than 20/60. CBS is also more likely to occur with binocular versus monocular disease [15]. By contrast, when release hallucinations occur in the setting of acute cerebral or optic nerve injury, they often occur concomitantly with the vision loss or after a latency of a few hours or days [18].

Associated auditory and/or tactile hallucinations are not consistent with CBS and suggest possible psychiatric disease, peduncular hallucinosis, narcoleptic hallucinations, or hearing or sensory loss. The images are typically colored and static, animated, or move en bloc across the visual field. The location of the hallucinations typically corresponds to the underlying vision loss, and as a result, can be monocular or binocular and/or restricted to one-half of the visual field. The content of the hallucinations usually does not have emotional impact or personal meaning for the patient who almost always recognizes them as unreal [5]. Despite this, a significant number of patients are distressed by their symptoms [19]. Hallucinations occur more often with the eyes open than closed, and patients often describe seeing the hallucinations when they are looking at a white background such as a wall, ceiling, or piece of paper. The hallucination will often disappear if the patient closes his or her eyes or looks away [13]. The most complex hallucinations occurred in the setting of sensory deprivation (dim lighting, inactivity, being alone) and in the evening or nighttime [7].

The duration of the hallucination ranges from less than one minute to continuous. Most patients report duration of several minutes. The frequency is variable; most patients experience hallucinations multiple times a day or week. Some patients experience only a few isolated episodes [4-8]. Associated symptoms depend upon the underlying disorder producing the visual loss. As examples, strokes involving the visual pathways produce visual field loss and sometimes other neurologic deficits, while macular degeneration and diabetic retinopathy produce loss of visual acuity without neurologic deficits [20]. Patients with CBS typically do not have an abnormal mental status or other neurologic deficits [13]. However, a syndrome of agitated delirium, visual hallucinations, and hemianopia can also be produced by lesions (usually stroke) affecting the medial aspect of the occipital lobe, the parahippocampal gyrus and the hippocampus [20].

The differential diagnosis of visual hallucinations include migraine aura, epilepsy, neurodegenerative disease, especially dementia with Lewy bodies and Parkinson disease, drugs, alcohol intoxication or withdrawal, metabolic encephalopathy, delirium, peduncular hallucinosis, narcolepsy and psychiatric disease. Anton's Syndrome is a rare medical condition is characterized by the patient going blind and not admitting to it. Other people become aware of this condition only after the patient has run or bumps into something and makes farfetched explanations for it. CBS is distinguished from these entities by the absence of other neurologic deficits and by the presence of known ocular disease.

Patients with new-onset visual hallucinations should have a complete neurologic evaluation screening for cognitive impairment, parkinsonism, and other neurologic deficits, and the medication list should be reviewed. Drugs like benzodiazepine, tricyclic antidepressants, narcotics, glucocorticoids are associated with visual hallucinations. In the absence of other neurologic abnormalities, and in the setting of known ocular disease (macular degeneration), further diagnostic evaluation may not be required.

By comparison, in the absence of known eye disease, a complete ophthalmologic evaluation with visual field testing should be performed. Brain magnetic resonance imaging is indicated if there is a visual field deficit or other focal neurologic deficit and for patients in whom the cause remains obscure [21]. Treatment is individualized according to the degree the patient is disturbed by their symptoms. While many find that symptoms have no significant impact on their life, up to a third of patients in one series reported that symptoms were distressing and had a negative effect on their quality of life [22]. Patients are often relieved to know that treatment is an option. Some patients can be taught to suppress the hallucinations by rapid eye movements from one target to another, especially away from the affected visual field, or blinking may temporarily suppress the hallucination [13]. Increasing arousal and visual stimuli by increasing illumination and reducing social deprivation are also reported to be useful for individual patients, but these techniques have not been systematically studied [21,23,24]. Patients with continuous hallucinations or those with disturbing imagery may need specific treatment. Treatment trials with low doses of atypical antipsychotics, cholinesterase inhibitors, serotonin reuptake inhibitors, or antiseizure drugs have anecdotal evidence of efficacy [25-27].

Age-related Macular Degeneration (AMD) is a degenerative disease of the central portion of the retina (the macula) that results primarily in loss of central vision. Central vision is required for activities such as driving, reading, watching television and performing activities of daily living. AMD is classified as dry (atrophic) or wet (neovascular or exudative) for clinical purposes. Dry AMD progresses to wet AMD in some patients. Findings in dry AMD may include subretinal drusen deposits, focal or more widespread geographic atrophy of the Retinal Pigment Epithelium (RPE), pigment epithelial detachments and subretinal pigment epithelial clumping. The pathogenesis of dry Age-related Macular Degeneration (AMD) is unclear. There is some evidence that abnormalities in components of Bruch's membrane and inflammation and chronic infection may also play a role. Early Age-related Macular Degeneration (AMD) is often asymptomatic. Patients with dry AMD may complain of gradual loss of vision in one or both eyes. This is often first noticed as difficulty reading or driving, scotomas, or reliance on brighter light or a magnifying lens for tasks that require fine visual acuity [28]. All patients should be encouraged to quit smoking or avoid initiating smoking due to the increased risk of progression to advanced age-related macular degeneration.

Patients with extensive intermediate-size drusen, at least one large drusen, or noncentral geographic atrophy in one or both eyes are treated with a daily oral eye vitamin supplement. Doses should be consistent with the Age-Related Eye Disease Study 2 (AREDS2) formulation (containing vitamin C 500 mg, vitamin E 400 international units, lutein 10 mg, zeaxanthin 2 mg, zinc 80 mg [as zinc oxide] and copper 2 mg [as cupric oxide]). The original AREDS formulation contained beta-carotene, which has been associated with an increased risk of lung cancer, particularly in smokers. Nonsmokers and others at low risk of lung cancer may use the original formulation, if available. Antioxidants have been hypothesized to prevent cellular damage in the retina by limiting the effects of free radicals produced in the process of light absorption [29-31].

Learning Points/Take Home Messages

- Charles Bonnet syndrome refers to a phenomenon of visual hallucinations occurring in patients with acquired vision loss affecting the eye or visual tracts in the optic nerve, chiasm, or brain
- The hallucinations of CBS may be elementary (unformed flashes of light or geometric shapes) or complex (formed images often of

animals, people, or scenes). These typically last a few minutes and usually recur frequently, daily or weekly

- The diagnosis is made when these hallucinations occur in patients with vision loss in the absence of psychosis, delirium, or other causes. When there is no known history of vision loss, evaluation should include toxicology screen, ophthalmology consult, neurologic evaluation, visual field testing and/or brain imaging
- Reassurance to patients is important and may be all that is required; however, treatment should be offered as well. Patients may be able to temporarily suppress hallucinations by closing their eyes or looking away
- Age-related Macular Degeneration (AMD) is the leading cause of adult blindness in industrialized countries. AMD is a degenerative disease of the central portion of the retina (the macula) that results primarily in loss of central vision. AMD is classified as dry (atrophic) or wet (neovascular or exudative)

Patient's Perspective

Since the diagnosis of my condition I feel more in control of my life. I have realized that due to my decreasing vision, I start seeing people. The doctors have reassured me that I have no mental problems and no stroke. My hallucinations have significantly decreased since the diagnosis of Charles Bonnet Syndrome and I have been taught eye exercise to suppress the hallucinations.

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