Charles Bonnet Syndrome in a Patient with Parkinson’s Disease and Bilateral Posterior Capsule Opacification

Abstract: Charles Bonnet syndrome (CBS) is a condition of visual hallucinations or disturbances occurring in patients with visual pathway pathology not due to underlying psychiatric, metabolic, or neurologic disease. A patient with Parkinson’s disease experiencing visual hallucinations was evaluated by the ophthalmology service and found to have decreased vision due to bilateral reversible posterior capsular opacification. The patient’s hallucinations did not improve on clozapine, a medication requiring careful monitoring due to potentially severe systemic side effects. However, the hallucinations resolved and vision improved after bilateral treatment of the posterior capsular opacification. Clozapine was then discontinued, and the patient was able to resume his previous Parkinson’s disease therapy. This case highlights the importance of considering visual pathway pathology as a contributing factor to visual hallucinations, even in patients with previously diagnosed underlying psychiatric, metabolic, or neurologic disease that could additionally be the etiology of the visual disturbances.

Keywords: visual hallucinations, clozapine, vision

Introduction

Charles Bonnet syndrome (CBS) is a condition of visual hallucinations or disturbances occurring in patients with visual pathway pathology. These are not due to underlying psychiatric, metabolic, or neurologic disease.1 The incidence of CBS increases with age and severity of vision loss.2 The differential diagnosis of CBS includes etiologies of dementia (vascular dementia, Alzheimer’s disease, etc.), psychosis (psychiatric disease, metabolic causes, etc.), Parkinson’s disease, and delirium. CBS is essentially a diagnosis of exclusion; therefore, other etiologies of hallucinations must be excluded.3 Although there is no specific and clearly agreed upon diagnostic criteria for CBS, the general required elements are (1) visual hallucinations, (2) vision loss, and (3) lack of confounding neuropsychiatric disorders causing visual hallucinations.4 There are many reports of CBS occurring in conditions that can cause severe vision loss such as macular degeneration and glaucoma.5 Additionally, there are reports of patients with relatively less severe and reversible vision loss also experiencing CBS and resolution after treatment.6–8 Table 1 highlights the case reports of CBS and the visual pathway pathology.

Nguyen8 previously reported a CBS case of a patient with visually significant bilateral posterior capsular opacification (PCO) experiencing hallucinations. After undergoing sequential treatment with neodymium-doped yttrium aluminum garnet
(Nd:YAG) capsulotomy in both eyes, the patient had an improvement in the perceived hallucinations. In this case report, we present a unique case of a patient with a baseline neurologic disorder also experiencing hallucinations in the setting of visually significant bilateral PCO.

**Case Report**

A 76-year-old white male with severe Parkinson’s disease leading to severe motor impairment and mild cognitive impairment was referred by to us by the neurology and psychiatry services for decreased vision affecting his ability to conduct his daily activities. He also reported seeing daily images of a little girl playing with blocks, a cat in his window, and men tearing down his neighbor’s roof that he recognized were not real. These visual disturbances were debilitating to the patient and made it difficult to complete work around his home and interact with family members. Prior to our evaluation, physicians from neurology and psychiatry had recently adjusted the patient’s pharmacotherapy in attempts to manage these visual disturbances thought initially to be due to the progression of his Parkinson’s disease. A trial of atypical antipsychotic therapy including gradually increasingquetiapine to 300 mg twice daily over the course of 3 weeks followed by the addition of pimavanserin 34 mg daily for 3 weeks failed to relieve his hallucinations. Most recently, his dose of carbidopa/levodopa was decreased, and clozapine was initiated and gradually increased to 150 mg twice daily for 3 weeks since the visual disturbances continued to be debilitating. Clozapine, in particular, is a high-risk antipsychotic medication with the potentially serious adverse effect of agranulocytosis along with possible metabolic, neurologic, and cardiac toxicities including hyperglycemia, hyperlipidemia, seizures, myocarditis, and pulmonary embolism. He continued to experience hallucinations while on clozapine when he presented to the ophthalmology service. His ophthalmic exam revealed a best-corrected visual acuity (BCVA) of 20/200 in each eye (OU). Ocular examination was remarkable for mild corneal guttate OU, posterior chamber intraocular lenses (PCIOL) OU, and visually significant PCO OU. The anterior and posterior segments were otherwise unremarkable. He underwent sequential Nd:YAG capsulotomy OU. At his one-month follow-up, the patient reported complete resolution of his hallucinations with BCVA of 20/25 in his right eye (OD) and 20/30 in his left eye (OS). The patient then resumed his previous carbidopa/levodopa dose and was tapered off clozapine with sustained resolution of the visual disturbances.

**Discussion**

Charles Bonnet Syndrome can occur in patient with both severe or nonsevere and both irreversible or reversible vision loss. Parkinson’s disease is a progressive neurologic disorder characterized by tremor, bradykinesia, and gait disturbances. Visual hallucinations are the most common psychotic symptom of Parkinson’s disease. These psychosis symptoms relating to Parkinson’s disease can be treated with a range of antipsychotics, including pimavanserin and clozapine, but overall, these therapies may not be effective and safe in all cases. We present a patient who experienced vision loss from posterior capsular opacification, a treatable cause of vision loss. With resolution of the patient’s visual hallucinations and disturbances after sequential Nd:YAG capsulotomy OU, the bilateral PCO was the apparent etiology of both the decreased visual acuity and visual disturbances, indicating CBS. Bilateral YAG capsulotomies resulted in improved visual acuity and complete resolution of his visual hallucinations. This case report demonstrates the importance

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**Table 1 Highlighted Case Reports of Visual Pathway Pathology Leading to CBS**

<table>
<thead>
<tr>
<th>Case Report</th>
<th>Etiology of CBS</th>
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<tbody>
<tr>
<td>Hashmi FK, Ogra S, Madge S. 2019</td>
<td>Upper eyelid ptosis</td>
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<tr>
<td>Kolarova H, Catarino CB, Priglinger C, Klopstock, T. 2019</td>
<td>Leber’s hereditary optic neuropathy</td>
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<tr>
<td>Kim US. 2018</td>
<td>Multifocal intraocular lens</td>
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<tr>
<td>Bismup M, Swiec-Zubilewicz A. 2016</td>
<td>Exudative age-related macular degeneration</td>
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<tr>
<td>O’Hare F, Bentley SA, Wu Z, Guymen RH, Luu CD, Auton LN. 2015</td>
<td>Retinitis pigmentosa</td>
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<tr>
<td>Lerario A, Ciammola A, Poletti B, Girotto F, Silani V. 2013</td>
<td>Orbital Pseudotumor</td>
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<tr>
<td>Hashemi N, Zhang J, Gelman R, Lee Ag. 2012</td>
<td>Pituitary adenoma</td>
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<tr>
<td>Khadavi NM, Lew H, Goldberg RA, Mancini R. 2010</td>
<td>Eye patching</td>
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<tr>
<td>Jackson ML, Drohan B, Agrawal K, Rhee Dj. 2011</td>
<td>Glaucoma</td>
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<tr>
<td>Ashwin PT, Tsaloumas MD. 2007</td>
<td>Occipital lobe infarction</td>
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<tr>
<td>Tan CS, Sabel BA, Au Eong KG. 2005</td>
<td>Enucleation</td>
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</table>
of a thorough ocular exam and treating any potential reversible visual pathway pathology to improve visual function to assist in decreasing hallucinations in CBS. This patient’s case, in particular, had a co-existing neurologic condition that can cause hallucinations, but with improvement in visual function by treating the PCO, the patient was able to be relieved of his hallucinations and taken off of anti-psychotic pharmacotherapy, which has the potential for serious adverse systemic events. A clear etiology explaining how PCO could lead to visual hallucinations is not known, but a prevailing theory for CBS is that any visual pathway pathology can lead to visual deafferentation and then cortical hyperexcitability within the occipital cortex. It is critical for providers to consider CBS as a diagnosis in patients with visual hallucinations or disturbances, especially those patients with an underlying confounding neurologic disorder. Referrals for ophthalmological evaluation may uncover diagnoses and etiologies of visual pathway pathology that may be treatable and assist in improving visual disturbances due to CBS.

Ethics Statement
Written informed consent was provided by the patient to have the case details published. Institutional approval was not required to publish case details.

Disclosure
The authors report no conflicts of interest in this work.

References