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Visual release hallucinations presenting as psychosis – a scoping review

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Review

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Abstract

Objective. Visual release hallucinations are perceptual disturbances that occur in individuals who have experienced vision loss. Almost 50 million people worldwide are believed to experience visual release hallucinations, yet they are profoundly underdiagnosed. Although first described within the Charles Bonnet syndrome, the paradigm underlying this syndrome precludes their consideration in many populations, such as those with underlying psychiatric illness or dementia. Consequently, visual release hallucinations have rarely been studied in patients presenting with psychosis. We conducted a scoping review to determine whether visual-release hallucinations occur in psychotic patients.

Methods. The PubMed research database was searched from inception through April 2023. Cases were collected reporting on psychotic patients experiencing suspected visual release hallucinations. Individual treatment courses and responses were extracted.

Results. Thirteen cases compiled from 11 different studies were summarized to provide baseline characteristics and overall trends in treatment response. Most patients did not remit from pharmacological management alone. All patients who received reafferentation therapy remitted, though many were not candidates. Almost half of the patients did not achieve remission.

Conclusions. Visual release hallucinations can manifest in psychosis and may contribute to treatment-resistant psychosis among psychiatric populations. A shift in our understanding of visual release hallucinations may aid their recognition in psychotic patients by shifting the focus toward visual release features. Recognizing release features among patients with hallucinatory conditions may open new treatment avenues for managing patients with psychosis. A preliminary screening index for visual release features is provided to support this shift.

Introduction

History

Visual release hallucinations (VRH) are neuropsychiatric phenomena that were first characterized under the Charles Bonnet syndrome (CBS).[1] In 1760, the Swiss naturalistic philosopher Charles Bonnet described the emergence of visual hallucinations following vision loss in his grandfather and later in himself.[2] The condition was not named until 1967 after a case series was published by De Morsier calling it the Charles Bonnet syndrome (CBS).[2] The diagnostic criteria for CBS have been debated ever since.[2,3] Even after 250 years, no formal guidelines are universally accepted for diagnosing CBS.[3] Suggested diagnostic criteria for the condition include age, type of hallucination, insight, and absence of the following – cognitive impairment, delusions, hallucinations in other sensory domains, psychiatric history, as well as hallucinogenic drug exposure.[3] The most common diagnostic criteria recognized for CBS are vision loss, the presence of formed-complex hallucinations, and preservation of insight, but even this set of criteria is rejected by more than half of clinicians.[3] Table 1 depicts the historical variability amongst CBS diagnostic criteria.

The conceptualization of CBS remains ambiguous.[3] Under most diagnostic schemas it does not recognize VRH that occur in psychotic patients or is comorbid with other hallucinatory conditions.[3] Consequentially, recognition of VRH in psychiatric populations is limited using the CBS paradigm. Given its ambiguity and anachronistic restrictiveness, the authors suggest we abandon the eponym of CBS. For clarity, "CBS" will be replaced with "VRH" henceforth.

Epidemiology

The lifetime prevalence of VRH in patients after vision loss is highly debated.[2] In one meta-analysis, VRH occurred in ~20% of cases with visual impairment, leading to an estimated global prevalence of 47 million people.[4] Still, estimates in other studies range from 0.4% to 30%.[2] There are many potential causes for this variability. One reason may be inadequate screening, as research has shown that patients rarely disclose release phenomena due to the stigma of being implicated as psychotic.[1] Another cause may be variability in diagnostic criteria. For example, some diagnostic paradigms do not allow for VRH in patients with dementia and neurocognitive

Table 1. Charles Bonnet syndrome criteria

	Inclusion			
Criteria	Included *N*	Total *N*	%	Details
Age (lower limit)	10	33	30	Ranged 18–65 years
Type of hallucinations	25	33	76	2/3rd mandated complex
Insight	18	33	55	1/3rd full insight, 2/3 partial or full
No other hallucinations	16	33	48	
No delusions	14	33	42	
No cognitive impairment	14	33	42	Not all studies tested cognition
No past psychiatric history	22	33	67	Diagnoses rarely provided
No past hallucinogenic use	13	33	39	Compounds not specified

disorders.[3] However, given that VRH directly correlate with cognitive impairment, social isolation, and low sensory environments they may be a common cause for nocturnal agitation in dementia termed "sundowning".[1] Whether or not dementia patients are included when estimating VRH prevalence may contribute substantially to inter-study variations.

To establish accurate prevalence estimates authors must not only consider all the populaces in which VRH occur but also their variable manifestations. VRH has traditionally been limited to complex hallucinations, yet evidence suggests they may be simple phenomena as well.[1-3] Simple hallucinations, which are also referred to as elementary or unformed; consist of photopsias, basic shapes, grid-like designs, and branching patterns.[2] Complex hallucinations are formed, vivid images of people, faces, vehicles, animals, plants, and objects.[2] Complex VRH is often qualified as "Lilliputian", meaning the image perceived is distortedly smaller than it would be experienced if witnessed directly from the external world.[5] Ultimately, VRH present with a spectrum of manifestations across many different populaces.

Pathophysiology

VRH are release phenomena that develop following sensory deafferentation.[6] Deafferentation is an interruption or destruction of an afferent nerve pathway.[6] This deafferentation may be transient or chronic and can occur due to damage at any point in the sensory pathway.[1,6] Consequentially, there are numerous conditions that provoke VRH.[1] Common pathologies include glaucoma, cataracts, myopia, diabetic retinopathy, macular degeneration, retinitis pigmentosa, optic neuritis, temporal arteritis, retinal vein or central retinal arterial occlusions, and cerebral/occipital infarctions.[1]

Following deafferentation, the brain undergoes several pathological changes. Single-photon emission computed tomography (SPECT) studies provide evidence that deafferentation is associated with hypo-perfusion of the primary and secondary visual cortices and hyper-perfusion of the striatum and thalamus.[6] Positron emission tomography (PET) imaging reveals similar patterns related to metabolic disturbances.[6] Functional magnetic resonance imaging (fMRI) studies have shown that visual deafferentation is associated with hyperexcitability in the visual cortex and salience network, and increased connectivity between the visual cortex and other cortical regions.[6] Increased activity of brain regions that correlate with the type of VRH are also described, for example, increased activity at the fusiform nucleus in patients experiencing VRH involving faces.[6] Collectively this research suggests that incoming afferent tone normally exerts an inhibitory effect on the sensory cortex.[6] Following deafferentation, cortical disinhibition and hyperexcitability allow for the provocation of release phenomena.[6] Whether release phenomena are provocations of latent hallucinations, exacerbations of pre-existing hallucinatory conditions or a sufficient independent risk factor unto itself for hallucinations remains unclear (Figure 1).

Management

Reafferentation therapy

Minimizing deafferentation is the standard treatment for VRH.[1,6] Initial strategies optimize residual vision via spectacles, contact lenses, optical aids, and/or low-vision rehabilitation.[3,6] Improving vision diminishes the frequency of VRH.[6] When feasible, reversal of deafferentation termed "reafferentation" is performed through surgical or medical interventions.[1,6] Therapeutic options can include excision of cataracts, diabetic retinopathy laser surgery, intravitreal ranibizumab for macular degeneration, etc.[6] Visual pathway restoration through reafferentation can often curatively resolve VRH.[1-3]

Pharmacological interventions

Medications for managing VRH vary.[1,3,6] Antipsychotic drug treatments yield mixed results clinically; nevertheless, second-generation agents such as quetiapine or olanzapine are commonly prescribed.[1,3,6] Their efficacy may be attributable to $5HT_{2A}$ receptor antagonism as these receptors are concentrated in the visual cortex and are thought to be instrumental in the development of visual hallucinations.[7]

Small studies also report efficacy with cholinesterase inhibitors (e.g., donepezil), antiepileptic agents (e.g., valproate, carbamazepine, gabapentin, or clonazepam), serotonin reuptake inhibitors (e.g., escitalopram or venlafaxine), and the $5\text{-}HT_4$ receptor agonist cisapride.[1]

Behavioral & psychological interventions

Behavioral interventions are sometimes effective in mitigating VRH.[1,2] Effective techniques include blinking, improving lighting conditions, rapidly moving eyes to concentrate on something outside the visual field &/or socializing with someone.[1,2] Enhancing patient acceptance of VRH has also been found to improve prognosis & gradually reduce symptoms.[6] Psychological interventions such as hypnosis, relaxation training, distraction therapies,

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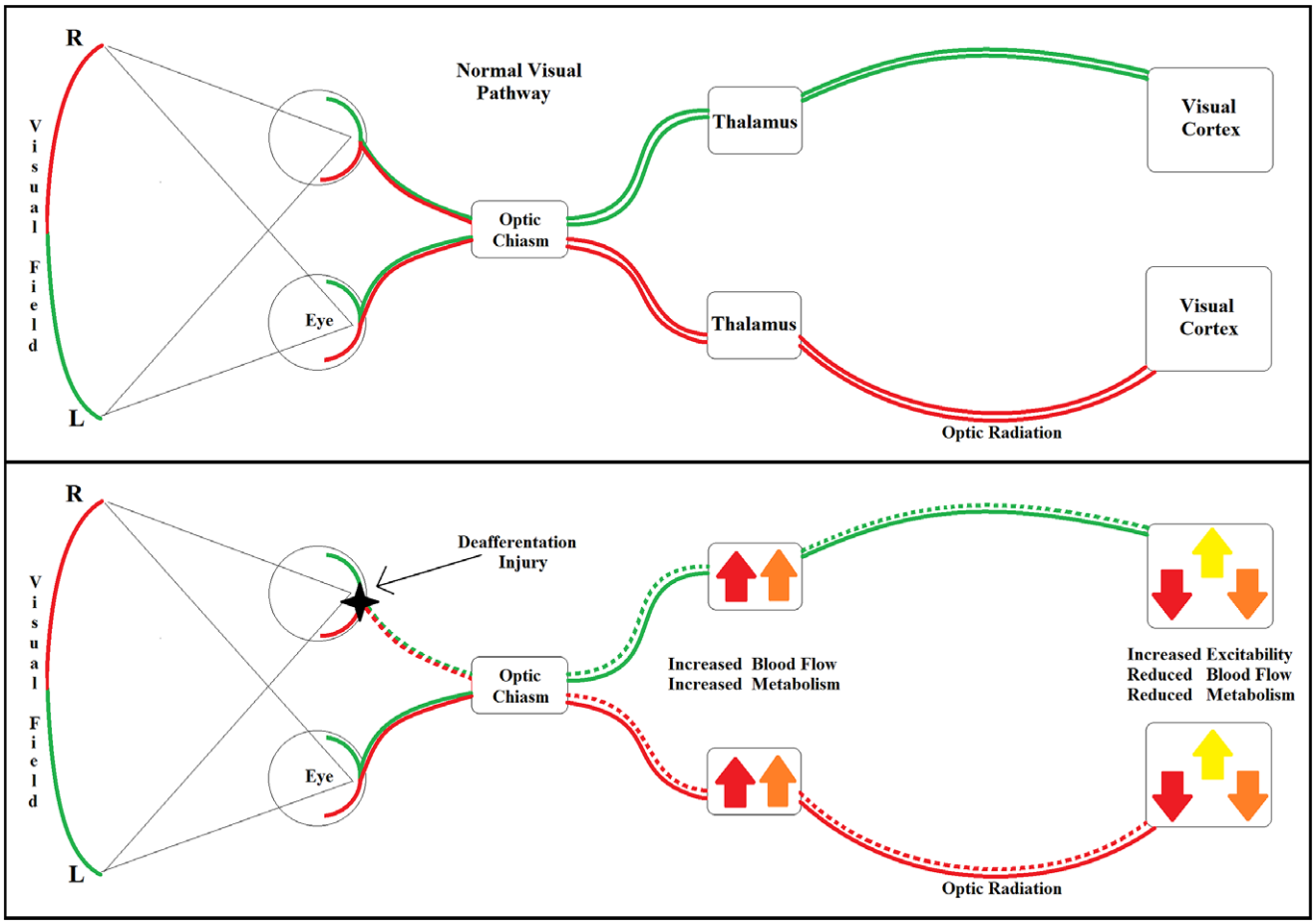

Figure 1. A simplified model for a deafferentation injury. The top diagram depicts a normal visual pathway, and the bottom depicts a visual pathway with a deafferentation injury secondary to ocular pathology. The solid lines indicate an intact sensory pathway, while the dotted lines indicate deafferentation. Changes in blood flow (red arrow), metabolism (orange arrow) and excitability (yellow arrow) are denoted.

cognitive remodeling, and psychotherapy have been shown to improve acceptance and coping with VRH.[1]

Neuromodulation

Enhancing inhibitory tone in the visual cortex through neuromodulation may hold promise for attenuating VRH. Repetitive transcranial magnetic stimulation (rTMS) may exert excitatory or inhibitory effects on specific target areas depending on the utilized technique.[8] Inhibitory rTMS can be provided by low-frequency rTMS (LF-rTMS) or continuous theta-burst stimulation (cTBS).[8] LF-rTMS has shown benefits for the management of some visual hallucinations and non-psychotic VRH.[9-11] However, the most promising treatment for VRH may be transcranial direct current stimulation (tDCS).[12] TDCS has already demonstrated level II evidence for reducing VRH specifically, and the portability of a tDCS device makes it an ideal tool for treating patients with VRH across a variety of practice settings.[12]

VRH & psychosis

Most research on VRH has focused on non-psychotic manifestations encompassed under the diagnosis of CBS. Thus, VRH presenting in psychotic patients has not been thoroughly investigated. VRH in non-psychotic patients responds sub-optimally to pharmacological intervention alone; however, it is unknown whether VRH in psychotic patients responds similarly. It is also unclear if VRH can be detected in patients presenting with psychosis given the nature of CBS is not geared to detect VRH in psychotic populations. This paper reviews cases of possible VRH in genuinely psychotic patients and proposes that future emphasis of VRH should focus on visual release features.

Methods

A literature review was performed using keyword-based queries in the PubMed electronic database. The aim of this review was to identify case reports and case series where patients presented with suspected VRH in the absence of insight into their unreality. The review was conducted on April 01, 2023, and the search terms were: "Bonnet Psychosis", "Charles Bonnet Plus", "Atypical Charles Bonnet", and "Release Hallucination". Retrieval was limited to humans and publications after 1952. Duplicate reports and excluded studies were eliminated. Articles were included if they presented case reports or case series of patients experiencing visual hallucinations with a history of confirmed or suspected visual deafferentation. Then, texts were checked for eligibility criteria. Reviewers also examined the reference lists of eligible manuscripts to identify other possible sources. The final article selection encompassed cases of psychotic patients suspected of experiencing VRH. Patients were suspected of experiencing VRH if they developed

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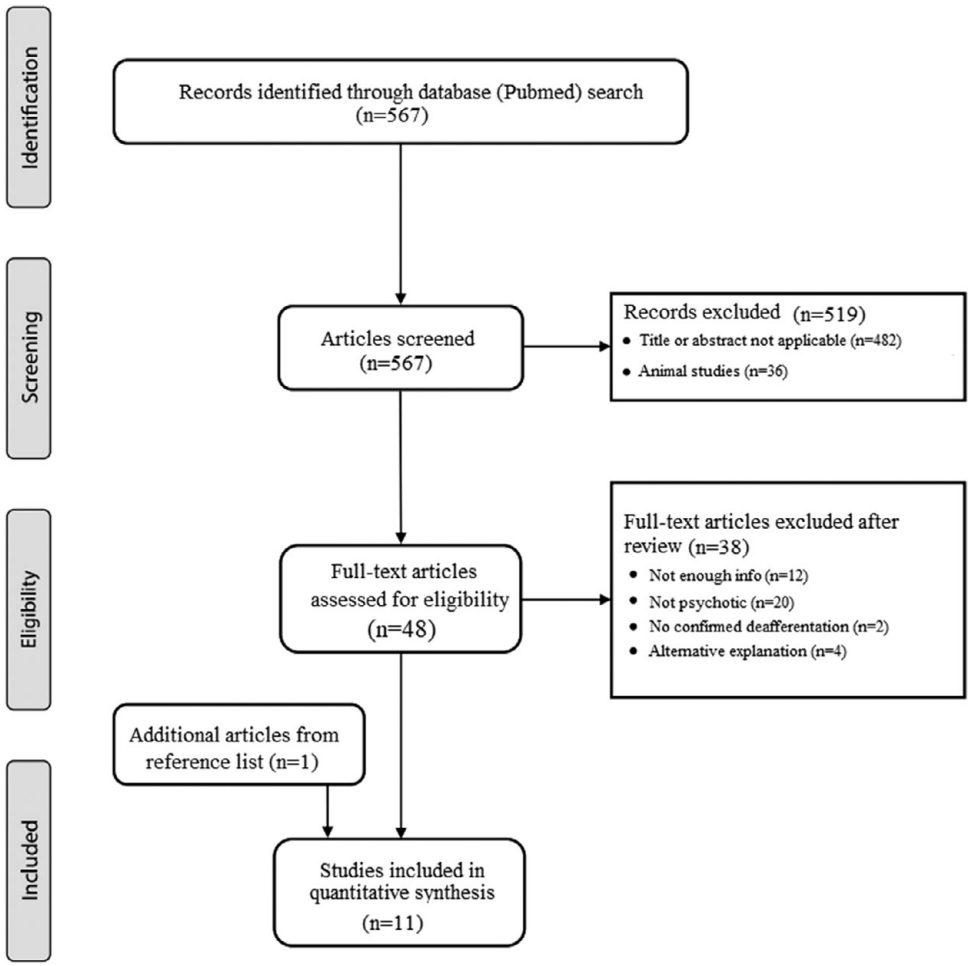

Figure 2. A search diagram showing the article selection and exclusion process.

Table 2. Visual release hallucination case highlights

Author	Age & sex	Deafferentation source	Types of visual hallucinations	Other psychiatric symptoms	Cognitive status	Pharmacological interventions	Reafferentation therapy	Response
Casey [13]	80 F	Age-related macular degeneration	Simple	Delusions	Normal	Haloperidol 5 mg	No	None
Casey [13]	85 M	Age-related macular degeneration	Simple Complex	Delusions	Impaired	Haloperidol 5 mg	No	None
Lanska [14]	9 M	Juvenile neuronal ceroid-lipofuscinosis	Simple Complex	None	Normal	None	No	None
Barnes 2001[15]	87 M	Age-related macular degeneration & cerebral infarction	Complex	Delusions	Normal	Sulpiride 200 mg	No	Full
Barnes 2001[15]	87 M	Age-related macular degeneration & cataracts	Complex	None	Normal	Sulpiride 200 mg	No	Full
Jackson and Ferencz [16]	69 M	Age-related macular degeneration	Complex	None	Unknown	None	Ranibizumab injections	Full
Makarewich and West [17]	75 M	Optic nerve infarction from temporal arteritis	Simple Complex	Delusions Tactile hallucinations	Unknown	Risperidone Rivastigmine	No	Partial
Arun et al. [18]	72 F	Cataracts & diabetic retinopathy	Complex	Delusions Auditory hallucinations	Normal	Aripiprazole 15 mg Risperidone 4 mg lorazepam 2 mg	Cataract excision	Full

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Table 2. *Continued*

Author	Age & sex	Deafferentation source	Types of visual hallucinations	Other psychiatric symptoms	Cognitive status	Pharmacological interventions	Reafferentation therapy	Response
Chatterjee et al. [19]	78 M	Cataracts	Complex	Depression	Normal	Escitalopram 10 mg Quetiapine 50 mg	Cataract excision	Full
Hill et al. [20]	90 F	Cataracts, retinal vein occlusion & vitreous hemorrhage	Complex	Delusions Auditory hallucinations	Impaired	Olanzapine 5 mg	Vitrectomy, phaco-emulsification & retinopexy	Full
Whitfield et al. [21]	78 M	Glaucoma, cataracts & sensory deprivation	Simple Complex	Delusions	Normal	Aripiprazole 10 mg	No	Partial
Maruzairi and Joo[22]	63 F	Postsurgical scarring from intraocular lens implantation	Simple Complex	Delusions Agitation	Normal	Quetiapine Zolpidem	No	Partial
Irizarry et al. [23]	42 M	Retinitis pigmentosa & traumatic brain injury	Complex	Delusions Agitation paranoia	normal	Olanzapine 15 mg Topiramate 300 mg Escitalopram 5 mg	No	Full

hallucinations for the first time following vision loss or if there was a notable change in hallucination frequency or quality following vision loss. These VRH were considered psychotic if insight into the falsity of these perceptual disturbances was persistently absent.

The exclusion criteria were as follows.

(1) The full article could not be obtained and the title or abstract contained insufficient information to determine if the patient is suspected of having experienced VRH.
(2) A review of the full article suggested that patient(s) consistently had at least partial insight into the falsity of their perceptual disturbances and were not psychotic.
(3) A review of the full article indicated that patient(s) had no confirmed visual deafferentation and therefore it was unclear if they were experiencing VRH.
(4) A review of the full article implied that patient(s) presentation may be better explained by another condition.
(5) The article did not contain patient-level data (Figure 2).

Once eligible studies were identified, relevant data was extracted and entered into a database. Items collected were age, gender, type of visual hallucinations, cause of visual deafferentation, other psychiatric symptoms, cognitive status, medications, reafferentation techniques, and response to treatment. Delusions were listed under other psychiatric symptoms only if they extended beyond absent insight into the patients' perceptual disturbances. Medication doses, if provided, are listed as the total daily dose. Clinical response was based on stated changes in VRH frequency. A "none" response meant that there was no notable change in hallucination frequency. A "partial" response indicated a reduction in frequency without complete disappearance. A "full" response inferred the complete resolution of VRH.

Results

In the initial search, 567 articles were identified, from which 519 were excluded. The remaining 48 papers were reviewed and 38 of these were removed. After searching the references of the remaining 10, an additional report was identified, providing 11 articles. All of them were case reports or case series, citing 13 individuals.

All cases either received pharmacological interventions, reafferentation therapy, or both. Table 2 summarizes the articles reviewed, including publication first author and year, patient age/gender, cause of visual deafferentation, visual hallucination type, other psychiatric symptoms, cognition, pharmacotherapies, reafferentation therapies, and treatment responses.

Data was analyzed to provide baseline epidemiological details and observations on treatment response by different methods. Table 3 summarizes the results obtained as follows. The mean age of psychotic patients with VRH was 70 years, with a median of 78 and a range between 9 and 90 years. Gender distribution was

Table 3. Summary of case findings

Category		Results		
		N	Total *N*	%
Sex	Female	4	13	31
	Male	9	13	69
Type of visual hallucinations	Simple	6	13	46
	Complex	12	13	92
Delusions	Present	9	13	69
Other sensory hallucinations	Tactile	1	13	8
	Auditory	2	13	15
Cognitive status	Provided	11	13	85
	Impaired	2	11	18
	Verified	3	11	27
Pharmacological response	None	4	11	36
	Partial	4	11	36
	Full	3	11	27
Reafferentation therapy response	Full	4	4	100

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Table 4. Preliminary visual release feature screening index (vRFI)

Visual release feature screening index (vRFI)			
Category	Subcategory	Details	Score
Deafferentation*	Subjective	Patient or family report subjective visual impairment but no observed deficits	1
	Objective	The patient has clear visual deficits on physical examination	2
	Confirmed	Trained professionals have diagnosed patients (Optometrist, Ophthalmologists, etc.)	3
Hallucination type	Complex	Fully formed (people, faces, animals, plants, objects, etc.)	1
	Simple	Unformed (photopsias, simple shapes, grid-like patterns, branching patterns, etc.)	1
Unique release phenomena	Lilliputian	Percept is shrunken relative to typical experience	1
	Clarity	Hallucinations have higher resolution and appear "more real" than typical vision	1
	Lateralization	Hallucinations only occur in one portion of the visual field predominantly	1
Other hallucinations	Absent	No auditory, tactile, olfactory or gustatory hallucinations	1
Insight*	Full	Insight is retained on all known occasions (no psychotic hallucinations)	1
	Partial	Insight is retained on at least some occasions	1
Delusions	Absent	No known or suspected delusional thoughts	1
Cognitive impairment	Present	Confirmed cognitive impairment	1
Psychiatric history	Absent	No known or suspected past or present psychiatric history	1
Substance use	Absent	No known or suspected past or present use of illicit substances	1
Hyperexcitability	Present	Electroencephalogram shows hyperexcitability of the visual cortex	1
Exacerbating factors	Low Sensory Environments	Hallucinations occur more frequently in low sensory environments (nighttime, dim lighting, quiet, etc.)	1
	Social Isolation	Hallucinations occur more frequently when socially isolated	1
Treatment responsive	Behavioral Interventions	Hallucinations occur less frequently following behavioral interventions (blinking, improved lighting, rapidly moving eyes, socializing, eyepatch application, etc.)	1
	Optimized Vision	Hallucinations occur less frequently following optimization of visual acuity (glasses, spectacles, contacts, etc.)	1
Reafferentation		Complete resolution of hallucinations following visual pathway reafferentation is DIAGNOSTIC	
Scoring instructions		Circle all scores that apply. Categories marked by asterisk (*) have mutually exclusive subcategories (pick only one option). Sensory deafferentation must be present to make a diagnosis of visual release hallucinations, all other categories are suggestive but non-essential	

9 males (69%) and 4 females (31%). Complex VRH were present in 12 cases (92%) and simple in 6 (46%); 5 cases (39%) reported both simple and complex VRH. Nine patients (69%) experienced delusions. Hallucinations in other sensory modalities were documented, with 2 patients (15%) experiencing auditory perceptions and 1 patient (8%) experiencing tactile hallucinations.

Most cases (85%) reported cognitive status but barely a quarter (27%) verified this with reliable neurocognitive testing. Only 2 patients (18%) had a known or suspected cognitive impairment, with 9 (82%) considered unimpaired. It should be noted that the mini-mental status exam (MMSE) was not deemed to be a reliable neurocognitive testing measure for this study.

Response to treatment was determined by the author's subjective reports. Responses to pharmacological agents were mixed: 4 (36%) denied improvement, 4 (36%) said there was some attenuation of VRH, and 3 (27%) reported full remission. All 4 subjects (100%) with visual reafferentation therapy achieved full remission. Overall, 7 patients (54%) achieved remission following treatment, leaving 3 (23%) with some progress, and 3 (23%) evidencing no improvement.

Discussion

VRH are a complex phenomenon whose prior study has predominantly focused on non-psychotic manifestations. While the original diagnostic criteria of CBS helped identify VRH thereby aiding investigations into their pathophysiology, this paradigm has limited ability to recognize VRH in psychotic patients. As this review shows, VRH likely contribute to psychosis in some populations. Still, though VRH may occur in psychotic patients, the validity of referring to these disturbances as VRH is not without problem. Even if a patient with a known hallucinatory condition develops vision loss and subsequently experiences a change in the nature or frequency of their hallucinations, who is to say that they are experiencing VRH rather than visual hallucinations with a multifactorial etiology?

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Many individuals who experience vision loss never go on to develop visual hallucinations.([2,5]) A modern synthesis of 8 models for complex visual hallucinations has argued that visual hallucinations may be a consequence of multiple factors, and their episodic nature is better understood by integrating various models into a unified framework rather than considering them in isolation.[24] Certainly, the episodic nature of VRH and its absence in many patients with profound vision loss highlights the importance of recognizing deafferentation as a part of the larger puzzle underlying visual perceptual disturbances.

However, this is not to undermine the importance of considering vision loss's specific impact on visual hallucinations. Further research must be conducted to elucidate how vision loss impacts hallucinatory experiences and psychosis across all populations. It is possible that visual perceptual disturbances may be uniquely changed in individuals experiencing vision loss, factors which the authors' term "visual release features". Identifying these features may aid the recognition of vision loss in new populations and help guide clinical management. Treatment-resistant psychosis is a growing problem in our society.([25]) With nearly 50 million people believed to experience VRH, under-recognition of deafferentation among patients experiencing visual hallucinations may be contributing to treatment resistance and suboptimal prognoses.([4]) Reafferentation therapy, alternative pharmacological interventions, and neuromodulation may offer better clinical outcomes in patients experiencing hallucinations with release features.

To aid identification of visual release features among psychiatric populations, a preliminary visual release feature screening index (vRFI) has been provided in Table 4. This screening index is currently under review at our institution for empiric validation. Hopefully, improved recognition of visual release features will improve clinical outcomes for managing patients with treatment-resistant hallucinations and psychosis.

Data availability statement. Additional data will be made available through correspondence with the corresponding author.

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