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Schizophrenia and Other Psychotic Disorders Among the Visually Impaired

Kamand Abedi, HBSc [1]*

[1] Department of Health, York University, Toronto, Ontario, Canada M3J 1P3

*Corresponding Author: abedikamand26@gmail.com

Abstract

Introduction and Definition: Schizophrenia spectrum and other psychotic disorders are characterized by delusions, visual and/or auditory hallucinations, disorganized thinking and speech, abnormal motor behavior, and negative emotional symptoms. Although the cause of these disorders remains unknown, hallucinations stand out as a prominent symptom. Historically, research on visual hallucinations in blind or visually impaired individuals has been limited. This entry aims to explore the existing data concerning the relationship between vision and symptoms of psychotic disorders, with an emphasis on visual hallucinations.

Current Research: Research indicates a correlation between schizophrenia and deteriorating eye health, likely stemming from prolonged visual hallucinations and the medications used to manage schizophrenia. Childhood blindness has been identified as a protective factor against psychosis, highlighting the role of glutamate receptors in the accuracy of prediction errors related to visual hallucinations. In older individuals with declining vision, Charles Bonnet syndrome stands as a notable exception, representing a cause of visual hallucinations linked to significant vision loss rather than a psychotic disorder.

Implications: There are no reported cases of schizophrenia among congenitally blind individuals. However, several studies on hallucinogenic drugs, such as lysergic acid diethylamide (LSD), in congenitally blind individuals have found that, rather than experiencing visual hallucinations, they report alterations in other senses, such as touch. This suggests that congenital blindness may serve as a protective factor against schizophrenia and other psychotic disorders, whereas late-onset vision loss does not confer the same protection.

Future Directions: Future efforts should prioritize destigmatizing psychotic disorders and promote more research on nonpsychotic disorders that present with visual hallucination symptoms, such as Charles Bonnet and Usher syndrome. This is particularly important given that many individuals with these disorders refrain from reporting their visual hallucinations due to fear of being misidentified as mentally ill and concerns about potential hospitalization.

Targeted Interventions: Research on the origins of visual hallucinations—whether they involve the brain, the eyes, or both—suggests that targeting the serotonin-glutamate system in therapy can improve visual hallucination symptoms in schizophrenia and other psychotic disorders. Furthermore, clinical interventions that target neurocognitive assessments help to individualize treatment based on personal symptoms and neural processing.

Keywords: schizophrenia; psychosis; vision; congenital blindness; visual hallucination

Introduction and Definition

Symptoms of Schizophrenia and Other Psychotic Disorders

Psychotic disorders constitute a category of mental illnesses characterized by symptoms including visual or auditory hallucinations, disorganized speech and thinking, negative symptoms such as emotional unresponsiveness to positive stimuli, lack of interest in social interactions, and abnormal motor behavior which may include catatonia—a reduced responsiveness to the environment resulting in temporary mutism, unusual body postures, or prolonged fixation on stimuli [1]. Schizophrenia, categorized under psychotic disorders, is distinct in that it primarily presents with visual or auditory hallucinations—seeing things not present or hearing voices externally [1]. Additional features specific to schizophrenia spectrum disorder include a diminished ability to display emotional expressions, often evident in difficulty maintaining eye contact or facial muscle stiffness, and a lack of motivation for self-initiated activities, leading to extended periods of inactivity [1].

Possible Causes

While the causes of Schizophrenia and other psychotic disorders are complex, research points to a combination of biological and environmental factors [2]. Biological factors

encompass a familial history of schizophrenia, prenatal stress, fetal malnutrition, infections, and complications during delivery leading to fetal hypoxia [2]. Genetic studies have identified specific proteins encoded in human genes, particularly DISC1 (disrupted in schizophrenia 1), as potential indicators of schizophrenia and psychotic disorder susceptibility [3]. Environmental influences, including urban living stressors like discrimination, dysfunctional family dynamics, and low socioeconomic status, also play a significant role [2].

Significance and Purpose

Research on schizophrenia and other psychotic disorders in visually impaired individuals has been limited, particularly regarding how visual symptoms manifest in blindness—a relatively new area of study [4]. This raises key questions: How do schizophrenia symptoms, especially visual hallucinations, present in blind individuals? Are they more prone to developing schizophrenia, or do they have an advantage? Do they experience visual or primarily auditory hallucinations? Additionally, do these hallucinations originate in the brain or the eyes? This entry reviews existing research on visual hallucinations and blindness to address these questions. It will explore whether experiences of those who are congenitally blind or who lost their sight later in life can illuminate the origins of visual hallucinations in psychotic disorders. It will also briefly discuss auditory hallucinations and the role of hearing in psychosis for blind individuals. Studying the effects of hallucinogens on blind individuals is crucial for understanding visual symptoms in this population. Insights into the role of vision in schizophrenia and other psychotic disorders are essential for developing interventions to destigmatize these conditions [5]. Differentiating between visual hallucinations stemming from ocular issues and those from the brain will help distinguish schizophrenia from conditions like Charles Bonnet syndrome, an age-related eye condition rather than a psychotic disorder [5].

Current Research

Visual Deprivation and Cerebral Changes

Computational psychiatry methods, a common technique used in studying the relationship between vision and psychosis, reveal that early-life visual deprivation leads to adaptations in the visual cortex [4]. Specifically, that levels of N-methyl D-aspartate receptor 2B (NR2B), a subunit of the N-methyl-D-aspartate (NMDA) receptor crucial for memory and learning, persist longer than N-methyl D-aspartate receptor 2A (NR2A), contributing to increased stability in higher-level priors [4]. Furthermore, the computational approach demonstrated that early visual deprivation induces plastic changes in the cerebral cortex, causing it to adapt and rely more on other sensory modalities [4]. The Bayesian Prediction Error Minimization Model further elucidated that in schizophrenia, mutations in glutamate receptor ionotropic N-methyl-D-aspartate 2B (GRIN2B) genes reduce NR2B function while increasing NR2A function [4]. This imbalance results in prediction errors that can manifest as visual hallucinations, as the brain falsely infers thoughts, movements, and emotions [4]. Therefore, a visually deprived brain, with enhanced NR2B function and precision in higher-level priors, may mitigate the risk of erroneous inferences, potentially acting as a protective barrier against schizophrenia and psychosis [4]. To explore whether blind individuals can experience visual hallucinations, LSD—a drug affecting 5-hydroxytryptamine 2A (5HT2A) serotonergic receptors known to induce visual hallucinations—was administered to blind subjects [4]. Congenitally blind individuals reported no visual hallucinations but did experience alterations in auditory perception, such as heightened auditory sensitivity [4].

Psychosis and Cognitive Changes

Other studies examining eye health in patients with schizophrenia and other psychotic disorders employ longitudinal methods to track deterioration over time. They utilize Electroretinography (ERG) to monitor changes resulting from prolonged visual hallucinations and assess the impact of antipsychotic drugs on the retina and other ocular structures [6]. Additional research investigates cognitive functions such as spatial awareness, memory, attention, auditory perception, and mental rotation tasks in congenitally blind subjects compared to a control group with normal vision but blindfolded. These studies aim to determine whether differences in cognitive performance exist between the two groups and if the absence of vision acts as a protective factor against psychosis compared to individuals with intact eyesight [7]. They also examine the relationship between visual functioning and psychosis, exploring abnormal perceptions of the world [7].

Non-Psychotic Disorders and Visual Hallucination

Case studies and magnetic resonance imaging (MRI) examinations of patients with Charles Bonnet syndrome, a condition characterized by visual hallucinations due to significant vision loss in the elderly [5], and Usher syndrome, a genetic disorder causing both visual and auditory degeneration, further contribute to understanding disorders with schizophrenia-like symptoms [4]. Charles Bonnet syndrome is an underreported condition associated with visual hallucinations due to age-related macular degeneration but lacks any other symptoms of psychotic disorders [8]. Patients with Charles Bonnet syndrome typically retain intact cognition but experience visual hallucinations due to declining and blurred vision, differing from psychotic disorders which involve alterations in both cognition and vision [8]. For instance, a 60-year-old man with significant visual impairment caused by autoimmune hypothyroidism reported visual hallucinations, but no pathological cause beyond declining vision was found [9]. Similarly, approximately 200 Parkinson's disease patients in nursing homes have

reported visual hallucinations without a psychological basis, which can often be attributed to old age rather than a mental disorder [10]. Additionally, Usher syndrome, a rare genetic disorder causing dual sensory impairment of vision and hearing, can also lead to auditory and visual hallucinations [11]. This condition, caused by gene mutations like retinitis pigmentosa, results in auditory hallucinations resembling thoughts as voices and visual hallucinations due to impairment of the retina [11].

Implications

Protective Factors against Schizophrenia

There are no reported cases of schizophrenia among congenitally blind individuals, though some cases of psychosis have been documented in those who became blind later in life, often manifesting as false auditory and sensory inferences rather than visual ones [4]. The absence of early visual input in congenital blindness may serve as a protective barrier against visual hallucinations, as these individuals lack prior visual cues for hallucinations to arise [4]. Congenitally blind individuals also show higher levels and functionality of NR2B protein receptors, which help mitigate prediction errors and false inferences of thoughts and emotions as visual hallucinations [4]. Prenatally, the human brain has its lowest NR2A/NR2B ratio, with NR2B peaking shortly after birth [4]. As development continues, NR2A levels increase to balance this ratio, coinciding with a child's exploration of their environment [4]. During critical periods of synaptic remodeling, the developing brain can become vulnerable, leading to reduced long-term potentiation or increased long-term depression due to NMDA receptor activation [4]. In visually deprived brains, the rise of NR2A may be delayed, allowing NR2B levels to persist longer [4]. This adaptation helps the visually deprived brain maintain stability and higher-level priors. Predictive coding theories suggest that abnormal prediction errors during development can lead to false inferences, manifesting as negative thoughts and hallucinations—core symptoms of schizophrenia [4]. This indicates that congenitally blind individuals may benefit from early visual deprivation, allowing their brains to adapt more effectively to higher-level priors during the NR2A/NR2B matching stage [4]. This adaptability enhances the brain's precision and stability regarding supramodal representations, reducing the risk of developing schizophrenia symptoms, such as delusions and hallucinations, thereby serving as a protective factor against the disorder [4].

The Role of Ocular and Cognitive Function in False Visual Outputs

To explore the relationship between eyes, brain, and visual hallucinations further, Electroretinography (ERG) studies of schizophrenia patients have shown increased glutamate release, a key neurotransmitter in retinal photoreceptors [6]. This glutamate increase, regulated by NMDA glutamate receptors, is linked to hypoactivity and dysregulation of NMDA receptors, leading to elevated dopamine release and resulting in visual distortions and hallucinations [6]. Particularly, individuals with schizophrenia without reported vision problems at birth often experience changes in eye structure and function [6]. These changes may include thinning of the retinal nerve fiber, widening of the retinal venule, and various dopaminergic and retinal abnormalities [6]. While the exact causes of these changes remain unclear, research suggests that they may be influenced by antipsychotic medications, the disorder itself, or other health conditions [6]. Factors that impact retinal function, such as psychotic disorders, can lead to heightened processing in the visual field, resulting in visual output that may appear noisy, intense, or degraded [6]. Additionally, Prolonged dopamine release, heightened glutamate output in the retina, and an imbalanced NR2A receptor ratio contribute significantly to deteriorating eye health in schizophrenic and psychotic patients, increasing their risk of developing conditions such as cataracts, strabismus, and poor visual acuity [6]. These symptoms, often observed in schizophrenia patients, may also coincide with declining intelligence quotient (IQ) levels in adolescence and widened retinal venules, potentially exacerbating psychotic symptoms by middle age [6]. Overall, the relationship between deteriorating eye health and worsened visual hallucinations suggests that they may be interchangeable [6].

Impact of Anti-Psychotic Drugs

Medications used to manage schizophrenia, particularly antipsychotic drugs, are implicated in the deterioration of eye health due to metabolic issues and diabetes among patients [6]. NMDA receptor abnormalities are prevalent in individuals with schizophrenia and are associated with increased dopamine release, which can lead to visual hallucinations [6]. However, research suggests that antipsychotic medi cations acting as NMDA antagonists, intended to manage these symptoms, may actually exacerbate visual hallucinations [6]. These drugs often contribute to metabolic issues, such as significant weight gain, in many patients [6]. The metabolic side effects of antipsychotic medications are evident in the high rates of diabetes among individuals with schizophrenia [6]. Diabetes, in turn, increases glutamate levels in the retina, further disrupting visual processing [6]. An example of such medications is ketamine, which functions as an NMDA antagonist but may cause retinal damage and cell death with prolonged use [6]. It is worth noting that while high rates of diabetes in schizophrenic patients may be influenced by antipsychotic drugs, diet also plays a significant role [12]. Individuals with schizophrenia and other mental health conditions often struggle with poor dietary habits, largely due to daily stressors [12]. Their diets typically contain high levels of carbohydrates and sugars, which can contribute to the development of diabetes and other metabolic issues [12].

The Role of Cognition in the Severity of Visual Hallucinations

Further studies assessing cognitive and visual processing in blind subjects reveal heightened auditory sensitivity among congenitally blind individuals and superior abilities in tasks involving localization, memorization, and processing of auditory information [7]. Despite similarities in cognitive experiences between congenitally blind and sighted individuals, some tasks requiring attention, adaptation, organization, filtering of irrelevant information, later alization, and imagery are performed better by sighted individuals [7]. Interestingly, these tasks have shown correlations with psychosis and visual hallucination severity in schizophrenia, suggesting that impairments in these tasks among congenitally and late-life blind individuals might serve as protective factors against such symptoms [7]. Research into the brains of blind individuals indicates that adaptations are independent of visual information and are not solely reliant on sensory modality [13]. Congenitally blind individuals demonstrate more independent information processing from visual stimuli compared to those who become blind later in life [13].

Visual Hallucinations among the Blind

Research on psychedelics and their effects on hallucinogenic experiences in blind individuals has shed light on visual hallucinations distinct from those in psychotic disorders [4]. A study found that no congenitally blind subjects reported visual hallucinations when administered LSD, while 13 late-blind subjects did [4]. Only one congenitally blind subject experienced non-visual hallucinations, emphasizing the role of vision in schizophrenia and psychosis [4]. A recent study suggests that individuals with their eyes closed do not share the same visual experiences as congenitally blind individuals [14]. Unlike them, sighted people perceive a color known as eigengrau in darkness, while pressure on their eyelids can produce geometric shapes and colors [14]. These findings highlight the optic nerve's role, which continues to gather faint visual information in sighted individuals, whereas this pathway is effectively blocked in those congenitally blind [14]. In a case study of a congenitally blind man who took LSD and psilocybin, he reported no visual hallucinations, but did experience altered auditory perception and difficulty detecting touch, describing faces as "melting" when touched [14]. The absence of visual hallucinations in congenitally blind individuals may result from the optic nerve's inability to transmit visual information to the brain [14]. This effectively acts as a protective barrier against visual hallucinations associated with psychedelics or psychotic disorders, as there is no visual data for the brain to misinterpret as visual hallucinations [14].

Future Directions

Certain illnesses can induce visual hallucinations resembling symptoms of schizophrenia and other psychotic [8]. Research indicates that many patients of Charles Bonnet syndrome hesitate to report their visual hallucination symptoms for fear of being misdiagnosed with a psychotic condition [5], and many elderly individuals in nursing homes experience visual hallucinations related to vision decline and Parkinson's disease, yet they are sometimes mistakenly medicated for mental disorders [10]. Individuals with Charles Bonnet syndrome, Usher syndrome, or Parkinson's disease who experience non-psychotic visual hallucinations may be reluctant to report their symptoms due to the social stigma associated with psychotic disorders such as schizophrenia and the fear of hospitalization [5]. Addressing the stigma surrounding psychotic disorders through public anti-stigma campaigns and increased education on mental health is crucial [15]. This approach promotes public safety, reduces fear, enhances accessibility to mental health services, and diminishes negative attitudes toward mental illness compared to physical conditions [16]. Future efforts should aim to raise awareness specifically among individuals affected by non-psychotic disorders that mimic schizophrenia symptoms, thereby encouraging them to openly discuss their hallucinatory experiences related to age and vision without fear of misinterpretation [5].

Targeted Interventions

Antipsychotic and psychedelic drugs both affect serotonin 5-HT(2A) and glutamate receptors, suggesting that targeting the serotonin-glutamate system may offer therapeutic benefits for schizophrenia [17]. While congenital blindness seems protective against schizophrenia, late-blind individuals may still experience hallucinations. For example, a congenitally blind man experienced LSD-induced synesthesia, highlighting sensory hallucinations and cognitive disruptions [18]. Acquired blindness from bilateral retinal detachment can lead to symptoms resembling schizophrenia, including paranoia and kinesthetic hallucinations [19]. Understanding the roles of different body systems, including vision, in visual hallucinations—whether originating in the brain or visual system—can aid in developing effective interventions for psychosis [19]. Overall, research on schizophrenia in visually impaired individuals is largely unexplored [4]. However, literature reviews indicate a connection between the eyes and brain in psychotic symptoms, with certain cognitive adaptations in congenitally blind individuals serving as protective factors [4]. Additionally, the severity of visual hallucinations may not be solely linked to visual or brain abnormalities but could also relate to NMDA antagonists—drugs used to alleviate psychotic features—or metabolic issues from dietary habits [6] [12]. Research provides insights for clinical practices targeting the root causes of visual hallucinations, whether from ocular problems, cognitive impairments, or medication complexities [6]. This individualized approach enables tailored interventions instead of a one-size-fits-all solution [20]. Clinical strategies

can include assessments of hallucination types, negative symptoms, and neurocognitive processes, which are essential for personalizing treatment [20].

List of Abbreviations
DISC1: disrupted in schizophrenia 1
ERG: electroretinogram
GRIN2B: glutamate receptor ionotropic N-methyl-D-aspartate 2B
5HT2A: 5-hydroxytryptamine 2A
IQ: intelligence quotient
LSD: lysergic acid diethylamide
MRI: magnetic resonance imaging
NMDA: N-methyl-D-aspartate
NR2A: N-methyl D-aspartate receptor 2A
NR2B: N-methyl D-aspartate receptor 2B

Conflicts of Interest
The author declares that she has no conflict of interests.

Authors' Contributions
KA: The sole contributor to the methodology and design of the article, analyzed and interpreted data from scientific literature, ensured the accuracy and validity of the literature, drafted the manuscript and the entire article with its subsections, and gave final approval of the version to be published.

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References
[1] Schizophrenia Syndrome and Other Psychotic Disorders. In: Diagnostic and Statistical Manual of Mental Disorders. 5th ed. Washington, DC: American Psychiatric Association; 2013. p. 87–8.
[2] Tsuang M. Schizophrenia: Genes and environment. Biological Psychiatry. 2000 Feb 1;47(3):210-20. https://doi.org/10.1016/S0006-3223(99)00289-9
[3] Ross CA, Margolis RL, Reading SA, Pletnikov M, Coyle JT. Neurobiology of schizophrenia. Neuron. 2006 Oct 5;52(1):139-53. https://doi.org/10.1016/j.neuron.2006.09.015
[4] Pollak TA, Corlett PR. Blindness, psychosis, and the visual construction of the world. Schizophrenia Bulletin. 2019 Oct 11;46(6):1418–25. https://doi.org/10.1093/schbul/sbz098
[5] Yacoub R, Ferrucci S. Charles Bonnet syndrome. Optometry - Journal of the American Optometric Association. 2011 Jul;82(7):421–7. https://doi.org/10.1016/j.optm.2010.11.014
[6] Silverstein SM, Rosen R. Schizophrenia and the eye. Schizophrenia Research: Cognition. 2015 Jun;2(2):46–55. https://doi.org/10.1016/j.scog.2015.03.004
[7] Landgraf S, Osterheider M. "To see or not to see: That is the question." The "protection-against-schizophrenia" (Pasz) model: Evidence from congenital blindness and visuo-cognitive aberrations. Frontiers in Psychology. 2013 Jul;4 :352. https://doi.org/10.3389/fpsyg.2013.00352
[8] Kester EM. Charles Bonnet syndrome: Case presentation and literature review. Optometry-Journal of the American Optometric Association. 2009 Jul 1;80 (7):360-6. https://doi.org/10.1016/j.optm.2008.10.017
[9] Lerario A, Ciammola A, Poletti B, Girotti F, Silani V. Charles Bonnet syndrome: Two case reports and review of the literature. Journal of Neurology. 2013 Apr;260: 1180-6. https://doi.org/10.1007/s00415-013-6857-7
[10] Sanchez-Ramos JR, Ortoll R, Paulson GW. Visual hallucinations associated with Parkinson disease. Archives of Neurology. 1996 Dec 1;53(12):1265-8. https://doi.org/10.1001/archneur.1996.00550120077019
[11] WU CY, CHIU CC. Usher syndrome with psychotic symptoms: Two cases in the same family. Psychiatry and Clinical Neurosciences. 2006 Oct;60(5):626-8. https://doi.org/10.1111/j.1440-1819.2006.01568.x
[12] Peet M. Diet, diabetes and schizophrenia: Review and hypothesis. British Journal of Psychiatry. 2004;184 (S47):S102-5. https://doi.org/10.1192/bjp.184.47.s102
[13] Ricciardi E, Handjaras G, Pietrini P. The blind brain: How (lack of) vision shapes the morphological and functional architecture of the human brain. Experimental Biology and Medicine. 2014 Nov;239 (11):1414-20. https://doi.org/10.1177/1535370214538740
[14] Tipado Z, Kuypers KPC, Sorger B, Ramaekers JG. Visual hallucinations originating in the retinofugal pathway under clinical and psychedelic conditions. European Neuropsychopharmacology. 2024 Aug;85: 10–20. https://doi.org/10.1016/j.euroneuro.2024.04.011
[15] Ertugrul A, Uluğ B. Perception of stigma among patients with schizophrenia. Social Psychiatry and Psychiatric Epidemiology. 2004 Jan;39:73-7. https://doi.org/10.1007/s00127-004-0697-9
[16] Rüsch N, Angermeyer MC, Corrigan PW. Mental illness stigma: Concepts, consequences, and initiatives to reduce stigma. European Psychiatry. 2005 Dec;20(8):529-39. https://doi.org/10.1016/j.eurpsy.2005.04.004

[17] Gonzalez-Maeso J, Sealfon SC. Psychedelics and schizophrenia. Trends in Neurosciences. 2009 Apr 1;32(4):225-32. https://doi.org/10.1016/j.tins.2008.12.005
[18] Dell'Erba S, Brown DJ, Proulx MJ. Synesthetic hallucinations induced by psychedelic drugs in a congenitally blind man. Consciousness and Cognition. 2018 Apr 1;60:127-32. https://doi.org/10.1016/j.concog.2018.02.008
[19] Marinho M, Moreira C, Catarina F. Psychosis in a blindness patient: A case report. European Psychiatry. 2016;33(S1):S532-S532. https://doi.org/10.1016/j.eurpsy.2016.01.1968
[20] Tortorella A. We should improve personalization of management in patients with a diagnosis of schizophrenia. Journal of Clinical Medicine. 2021 Dec 30;11(1):184. https://doi.org/10.3390/jcm11010184

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