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Casting shadows of perception: An exploration of visual hallucinations

By Pauline Stoltzner, PhD, MSN, APRN, FNP-BC, PMHNP-BC, CSAP and Cameron Duncan, PhD, DNP, APRN, FNP-C, PMHNP-BC, FAANP

Abstract: Hallucinations can be caused by biological, psychological, neurological, ophthalmological, and environmental factors. This article discusses a selection of the various conditions that can present with visual disturbances and hallucinations including schizophrenia, HIV, neurosyphilis, hyperammonemia, migraine, substance use, brain tumors, sleep disturbances, thyroid disorders, delirium, ophthalmologic conditions, and Lewy body dementia, providing an overview of the differential diagnosis of visual hallucinations. The mechanisms by which these conditions can lead to hallucinations are also discussed, and insight into the recommended medical workup for each is provided.

Keywords: assessment, behavioral health, differential diagnosis, hallucinations, mental health, neurologic mechanisms, visual disturbances, visual hallucinations

Due in part to the shortage of mental health care providers across the US, patients often first present to their primary care provider (PCP) with mental health symptoms. Though it is difficult to approximate due to underreporting of related symptoms, the prevalence of past-year hallucinations is estimated at 3% to 7% of people in the US.[1] Hallucinations are perceptions of an event or object that involve one or more of the five senses despite the absence of corresponding external stimuli. Hallucinations can be complex in that they may involve interactions among biological, psychological, neurological, and environmental fields. In this article, the authors explore visual hallucinations in adults to assist PCPs in their ability to understand, evaluate, and intervene in the presence of this symptom.

Understanding visual hallucinations

According to the *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition, Text Revision* (*DSM-5-TR*), "hallucinations are perception-like experiences that occur without an external stimulus." They are further described as being "vivid and clear" and "not under voluntary control."[2] It is unclear whether a single underlying mechanism causes visual hallucinations or if different mechanisms are associated with specific disease states.[3] No matter the circumstances, understanding the underlying cause of visual hallucinations is essential to proper management of the symptom.

Evaluating visual hallucinations

A common assumption is that presence of visual hallucinations indicates some type of psychotic disorder. However, it is important to recognize that there are many possible causes of hallucinations. Therefore, it is essential for the PCP to assess the patient fully to be able to adequately assess and address the symptom and underlying condition. This section explores some of the possible causes of visual hallucinations. It is important to note that the differential diagnosis for visual hallucinations is very broad, and this article provides an overview of a selection of possible etiologies. *Table 1* outlines the differential diagnosis as well as the corresponding lab and imaging orders that should be considered to further assess for each possible condition.

Psychiatric disorders

Patients with a psychiatric disorder, such as schizophrenia or bipolar disorder, may present with reports of visual hallucinations. According to the *DSM-5-TR* criteria for schizophrenia, in the setting of at least 6 months of symptomatic disturbance, at least two of the following symptoms must have been present continuously during a 1-month period for diagnosis: hallucinations, delusions, disorganized speech, grossly disorganized behavior, or negative symptoms such as anhedonia, avolition, and blunted affect.[2] Moreover, of these symptoms, a person must experience at least one of the following specifically for diagnosis: hallucinations, delusions, or disorganized speech. Finally, to be diagnosed with schizophrenia, a person must experience a marked disturbance in their level of functioning in terms of self-care, social factors, or occupational factors since the onset of the aforementioned symptoms. The onset of schizophrenia is often seen in the late teenage years to early 20s for men and late 20s to early 30s for women; it is considered early onset if diagnosed before age 18 years, with the disorder arising extremely rarely in individuals younger than 13 years.[4]

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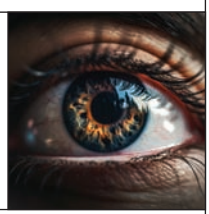

Patients experiencing an episode of mania or depression in a bipolar mood disturbance may also experience visual hallucinations. In the case of mania, the visual hallucinations are often accompanied by erratic behavior, a decreased need for sleep, racing thoughts, impulsive or dangerous decisions, excessive spending, and/or a decreased ability to care for oneself. Conversely, in a severe depressive episode, patients may report positive symptoms such as hallucinations, delusions, and disorganized speech and thoughts. Therefore, the PCP should assess for possible symptoms of depression as outlined by the SIGECAPS acronym: Sleep disturbances, Interests (anhedonia), Guilt/feelings of worthlessness, Energy levels, Concentration, Appetite, Psychomotor agitation or retardation, and Suicidality.[2] It is vital to include a comprehensive assessment of these criteria, including an evaluation of suicidal thoughts. In summary, to rule

out a mental health disorder as a cause for visual hallucinations, an assessment of family history, symptom onset, mood, thought processes, and accompanying behaviors must be undertaken.

HIV

HIV can invade the central nervous system (CNS) early in the course of the disease, during the first few days to weeks of primary infection, and it is reported that nearly 25% of patients with primary HIV infection will have symptoms of aseptic meningitis.[5] Additionally, psychiatric or cognitive effects can occur later in the disease course. HIV-associated neurocognitive disorder is a CNS disorder caused by HIV; it can occur in individuals with HIV who are untreated, inadequately treated, or in whom the treatment does not adequately control the infection within the CNS. When evaluating patients with suspected HIV, PCPs should perform a thorough history and physical exam, paying particular attention to symptoms that could suggest meningoencephalitis or other neuropsychiatric symptoms, such as hallucinations, delusions, or other cognitive, motor, or behavioral abnormalities. The subjective assessment should include a detailed review of the patient's past medical history, current medications, and any risk factors for HIV, including drug use and unprotected sex. The objective assessment includes monitoring for signs and symptoms of infection, including fever, cough, body aches, headaches, nasal congestion, neck stiffness, seizures, and sore throat, as well as completing a full neurologic exam.

Neurosyphilis

Syphilis is a sexually transmitted bacterial infection that is categorized into the following stages: primary, secondary, latent, and tertiary. If untreated, syphilis can progress to the tertiary stage, typically over decades but potentially much more quickly. Tertiary syphilis is very serious and is the most destructive stage. Neurosyphilis results from the syphilis bacterium crossing the blood-brain

Table 1. Recommended testing during workup for visual hallucinations

Suspected condition	Select potentially indicated tests
Schizophrenia or schizoaffective disorder	CBC count, CMP, urine drug screen, lipid panel, STI testing*, lumbar puncture, brain MRI
HIV	CBC count, CMP, urine drug screen, lipid panel, STI testing*, lumbar puncture
Neurosyphilis	CBC count, STI testing*, lumbar puncture
Hyperammonemia	Ammonia level, hepatitis panel, CBC count, CMP
Migraine	CBC count, CMP, EEG, brain MRI
Substance use	CBC count, CMP, urine drug screen, STI testing*
Brain tumor	Head/brain CT/MRI, CBC count, CMP
Sleep disturbance	CBC count, CMP, urine drug screen, EEG, sleep study, cortisol level, TSH
Thyroid disorder	TSH, free T3, free T4, TSI, TgAb, TPOAb, thyroid US, radioactive iodine uptake test
Delirium	Urine drug screen, head CT, urinalysis and urine culture, blood culture, CBC count, CMP, arterial blood gas, chest X-ray
Ophthalmologic condition	CBC count, CMP, A1C, lipid panel, brain MRI
LBD	MMSE or MoCA, brain MRI, brain PET, other tests as listed above for other conditions to rule out alternative potential causes
Medication adverse reaction	Testing will depend on the specific medication but may include CBC count, CMP, drug level testing if available, and testing to rule out other possible conditions.

Abbreviations: A1C, hemoglobin A1C; CBC, complete blood cell; CMP, comprehensive metabolic panel; CT, computed tomography; EEG, electroencephalogram; LBD, Lewy body dementia; MMSE, Mini-Mental State Examination; MoCA, Montreal Cognitive Assessment; PET, positron emission tomography; STI, sexually transmitted infection; T3, triiodothyronine; T4, thyroxine; TgAb, thyroglobulin antibodies; TPOAb, thyroid peroxidase antibodies; TSH, thyroid-stimulating hormone; TSI, thyroid-stimulating immunoglobulin; US, ultrasound.

*STI testing should include at least rapid plasma reagin (RPR), hepatitis panel, HIV, chlamydia (urine), and gonorrhea (urine).

Note: Table is not a comprehensive list of all possible tests that may be considered. Additionally, not all tests listed will be indicated in all situations.

barrier and causing infection of the meninges, the spinal cord, and/or the brain itself; this can occur at any stage of the disease.[6] The PCP should conduct a thorough subjective assessment of the patient's symptoms, which, in the case of neurosyphilis, may include visual hallucinations, depression, irritability, confusion, and other psychiatric, neurologic, or cognitive symptoms. The PCP should also perform a complete physical exam, including a neurologic exam, to assess for any neurologic deficits.

Hyperammonemia

Ammonia is a byproduct of a person's gastrointestinal tract made through a normal metabolic process and bacterial degradation of nutrients. In a healthy individual, the liver breaks down the ammonia into urea, which is then excreted by the kidneys. A number of medical conditions, including liver failure, cause the liver to function less than optimally, resulting in excess ammonia in the serum, which can cause acute encephalopathy, psychosis, hallucinations, and memory impairment. The effects of hyperammonemia are due to neuronal damage from oxidative stress, excitotoxicity, mitochondrial impairments, and inflammatory response, which can increase the glutamate levels in the brain.[7] When assessing a patient for possible hyperammonemia, the PCP should evaluate for a range of symptoms related to acute encephalopathy, psychosis (including hallucinations), memory impairment, and other associated clinical manifestations.

Migraine with aura

A migraine is a severe headache that can last for hours to days. The exact mechanism by which migraine attacks occur is unknown, but the condition is thought to be due to abnormal activity among the nerves, blood vessels, chemicals, and brain.[8] Auras, which occur before an impending migraine attack in some individuals, can include visual hallucinations, possibly arising from inappropriate activation of visual cortex neurons. When a patient presents with symptoms of severe headaches, the PCP should conduct a workup to determine if the cause is migraine. This workup should involve a thorough subjective assessment of the patient's symptoms including duration, frequency, and intensity of the headaches as well as any accompanying symptoms such as nausea, vomiting, and sensitivity to light and/or sound. The PCP should also complete a neurologic exam and rule out other causes of the headaches. Of note, an aura can occur before a seizure in some individuals; however, discussion of this situation is outside the scope of this article.

Substance use

Numerous drugs and illicit substances can result in visual hallucinations. This article cannot provide a summary of each drug associated with visual hallucinations; however, an abbreviated and incomplete review of this expansive subject is included here. The most common drugs known to cause visual hallucinations are amphetamines, scopolamine, phencyclidine, ketamine, lysergic acid diethylamide (LSD), and psilocybin.[9] Amphetamines increase dopamine in the synaptic cleft, partly due to inhibition of dopamine reuptake, and lead to excessive glutamate release in the cortex. Scopolamine increases acetylcholine levels in the substantia nigra and ventral tegmental area, thereby increasing striatal dopaminergic activity. Phencyclidine and ketamine work similarly to amphetamines in that they increase dopamine in the brain but differently in that they are N-methyl-D-aspartate (NMDA) receptor antagonists and thus cause decreased excitability of neurons. The specific mechanism by which LSD causes visual hallucinations is not fully understood, but it is believed that the drug's agonist action on the 5-HT_{2A} receptors of the thalamus increases glutamate levels in the corticocortical and corticosubcortical transmissions and increases glutamate release into the prefrontal cortex.[9] Similar to LSD, the exact mechanism that causes visual hallucinations with use of psilocybin is unknown. However, it is known that psilocybin binds to and activates 5-HT_{2A} receptors and causes hallucinogenic effects when the dose is greater than 15 mg.[10] Although effects on the brain and signs and symptoms vary widely depending on the specific drug and its mechanism of action, PCPs should be attentive to various potential indicators of substance use, such as dilated pupils, sweating, or tremors, and of indicators of I.V. drug use,

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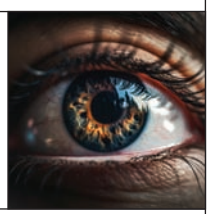

such as scarring or markings along the veins in the arms or legs. Additionally, the subjective assessment should include a thorough health and social history, with inquiry about use of any recreational drugs, holistic herbs, or other substances and about the overuse of any prescribed medications.

Brain tumors

Brain tumors, whether benign or malignant, can cause hallucinations. A study in 2019 found that hallucinations connected to brain tumors may be due to the neural network and connections among the cerebellar vermis, inferior cerebellum, and the right superior temporal sulcus. The location of the tumor in the brain affects the type of hallucination that may occur. For instance, lesions connected to the lateral geniculate nucleus in the thalamus can cause visual hallucinations.[11] When assessing for a potential tumor, providers should inquire about possible signs or symptoms of neurologic involvement such as personality changes, motor deficits, and speech or memory problems and should conduct a thorough neurologic exam, including assessment of cranial nerve function, motor coordination, reflexes, and sensory perception, looking for any focal deficits or abnormalities that could indicate the location of the tumor. Additionally, fundoscopy should be performed and visual acuity and visual fields assessed to evaluate for any optic nerve involvement. Further, providers should assess mental status, level of consciousness, memory, and cognitive function.

Sleep disturbances

During a period of sleep, the body rests and repairs its systems, including the brain. After 24 hours without sleep, the body will attempt to flood the brain with the sleep hormone melatonin and different ions that either polarize or depolarize the neurons.[12] After 48 hours without sleep, the neurons become more depolarized, causing an individual to be more alert, excitable, and awake; this can result in hypervigilance, irritability, visual hallucinations, delusions, and/or paranoia.[13] Each subsequent day that a person is awake without sleep, the hyperactivity in the brain worsens, resulting in exacerbated symptoms. PCPs should inquire about the patient's normal sleep routine, including what time they go to bed, what time they wake up, and the number of hours they spend in bed, as well as about difficulty with sleep initiation, sleep maintenance, early morning awakenings, restfulness of sleep, and daytime sleepiness or fatigue. The patient should be encouraged to keep a sleep diary that includes what time they went to sleep and woke up and how rested they felt when they woke up. The possibility of mania in bipolar disorder or substance use should be considered when an individual does not sleep for days at a time.

Thyroid disorders

Hyperthyroidism has the potential to cause neurologic or psychiatric symptoms, including visual hallucinations. Numerous theories speculate on the pathogenesis of these symptoms including excessive local production of the thyroid hormone triiodothyronine (T3) in the hippocampus and cortex of the brain; increased sodium current density in the hippocampal region caused by thyroid hormones, thereby causing neuronal excitability; and changes in the neurochemistry of and cerebral metabolism in the limbic system.[14]

Additionally, Hashimoto encephalopathy, a rare condition involving elevated levels of thyroid autoantibodies, such as thyroid peroxidase and antithyroglobulin antibodies, is associated with neuropsychiatric symptoms. This condition can occur in patients with subclinical hypothyroidism, hypothyroidism, or hyperthyroidism, as well as in patients who are euthyroid.[14] In Hashimoto encephalopathy, thyroid autoantibodies can bind to astrocytes in the cerebellum and to cortical neurons, potentially creating inflammation and various severe symptoms such as visual hallucinations, delusions, coma, and seizures.[15]

Providers should elicit a detailed history, including for existing thyroid conditions, thyroid-related treatments or surgeries, and family history of thyroid conditions, and should ask about any symptoms that may be associated with hyperthyroidism, such as unexplained weight loss, heat intolerance, sweating, palpitations, tremors, anxiety, and difficulties with sleep. Physical assessment should include measurement of BP and pulse and palpation of the thyroid.

Delirium

Delirium is a reversible neuropsychiatric condition that can occur in individuals of all age groups but is especially troublesome for older adults due to its potential negative outcomes, especially when detection is delayed. Symptoms of delirium usually have a quick onset, beginning

over a few hours to a few days.[16] Delirium often presents with visual hallucinations, delusions, and significant confusion. It can occur due to various underlying causes, such as urinary tract infections, sepsis, metabolic abnormalities, hospitalization, and adverse reactions of medications. Factors such as electrolyte disturbances, hypotension, and bacteremia may contribute to the development of delirium. If an underlying cause can be identified and treated promptly, any psychosis and visual hallucinations may be totally reversible.[17]

Assessment should encompass a thorough subjective and objective assessment including cognitive testing and use of a delirium assessment tool, medication history, and a comprehensive medical evaluation to identify the underlying cause of delirium and develop an appropriate treatment plan. Providers should inquire about urinary symptoms such as dysuria, urinary frequency, odor, hesitancy, retention, incontinence, or other changes in urinary habits, as well as about symptoms of other common delirium-precipitating conditions. Physical assessment should include measurement of vital signs. The provider should assess for changes in behavior, attention, and level of consciousness.

Ophthalmologic conditions

Eye floaters, also known as vitreous floaters, are collagen fibers that clump and cast shadows on the retina, resulting in visual dark spots, squiggly lines, or cobweb-like structures that appear to float over the visual field and that are common in people over the age of 50 years.[18] It is important to include this condition in the differential diagnosis because patients may occasionally present with eye floaters and mistakenly refer to them as visual hallucinations due to unfamiliarity with the appropriate medical terminology to describe this phenomenon. Patients may describe them as "seeing things" or experiencing visual disturbances, which providers could interpret as presence of visual hallucinations. It is important to note, however, that the pathophysiology of eye floaters is distinct from hallucinations associated with psychiatric or neurologic conditions. Other ophthalmologic conditions to consider that may cause visual disturbances but do not cause visual hallucinations include retinal tear and detachment.

Charles Bonnet syndrome (CBS) is characterized by simple or complex visual hallucinations in patients who have partial or complete visual loss. It is thought to be caused by increased activity in the visual cortex following reduced sensory input from the eyes, potentially resulting in the brain attempting to fill in the visual gaps from the impaired vision. This mismatch of activity creates visual experiences that are not grounded in reality. These hallucinations are exclusively visual in nature, usually temporary, and are not related to behavioral or mental disorders.[19] Whereas eye floaters are a result of physical changes within the eye, CBS stems from alterations in the brain's perception and processing of visual information.

To determine if the ophthalmologic symptoms the patient is experiencing are benign or a sign of something more serious, a detailed history, including when the visual hallucinations started, how long they last, as well as the degree and type of impact they have on the patient's daily life should be conducted, in addition to a comprehensive eye exam. Physical assessment should include evaluation of visual acuity with the Snellen eye chart and a thorough neurologic assessment to evaluate cognitive function, cranial nerve function, motor coordination, and reflexes. Various tests should be performed to rule out any metabolic or structural processes.

Lewy body dementia

Lewy body dementia (LBD) is a complex neurodegenerative disorder more commonly found in older adults that involves the presence of Lewy bodies, composed primarily of misfolded alpha-synuclein protein, in the brain. LBD encompasses two closely related conditions: dementia with Lewy bodies and Parkinson disease dementia. A signature trait of early-stage LBD is visual hallucinations, which can significantly impact a patient's cognition and quality of life. The pathophysiology of visual hallucinations in LBD is multifactorial and not yet fully understood. Lewy bodies are believed to interrupt brain signaling, especially among the acetylcholine and dopamine systems, and to play a big part in causing hallucinations. One study found that when visual hallucinations occur within the first 5 years of dementia onset, it is five times more likely to be from LBD than

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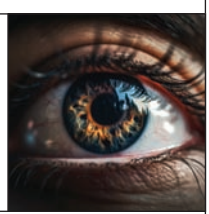

Table 2. Treatments for the underlying causes of visual hallucinations

Underlying condition	Treatment approach
Schizophrenia or schizoaffective disorder	• Consultation with a psychiatric specialist (for example, a PMHNP) is crucial for treatment decisions. • Antipsychotics are the mainstay of treatment. Choice of antipsychotic depends on specific subtype.
HIV	• Refer to infectious diseases specialist. • Treatment involves antiretroviral therapy. • Secondary infection prevention is essential. • Prompt detection and treatment of HIV-related cognitive, neurologic, or behavioral signs or symptoms can enable treatment initiation or adjustment, leading to improved outcomes.
Neurosyphilis	• Refer suspected cases to infectious diseases specialist. • Recommended treatment involves an extended course of I.V. antibiotics. • Early recognition and treatment can prevent further neurologic damage and improve patient outcomes.
Hyperammonemia	• Treatment depends on specific cause. • Possible interventions may include dialysis and/or use of medications such as lactulose and rifaximin.
Migraine	• Various pharmacologic treatment options are available for prevention and/or treatment including NSAIDs, triptans, CGRP inhibitors, select beta-blockers, amitriptyline, venlafaxine, select antiepileptics, onabotulinumtoxinA, and others. • Nonpharmacologic prevention and treatment options should be included in plan of care.
Substance use	• Acute and long-term treatment will vary depending on specific substance.
Brain tumor	• Refer to appropriate specialists, which include oncologists, neurosurgeons, and neurologists.
Sleep disturbance	• Sleep hygiene education is crucial. • Various pharmacologic treatment options are available for insomnia, including select sedative-hypnotics (zolpidem, zaleplon, eszopiclone), benzodiazepines (temazepam, triazolam), melatonin receptor agonists (ramelteon), and orexin receptor antagonists (suvorexant, lemborexant, daridorexant). Of note, many are intended only for short-term use. • Various other drugs are commonly used off-label for insomnia.
Thyroid disorder	• Treatment depends on the specific thyroid condition. • Treatment of hyperthyroidism may involve antithyroid medication, thyroidectomy, or radioactive iodine ablation. • Treatment of Hashimoto encephalopathy may involve glucocorticoids and/or other interventions.
Delirium	• Resolution of delirium may occur with treatment of underlying condition. • If antibiotics are indicated for an underlying infection, antibiotic selection should be tailored to the causative bacteria. • Visual hallucinations in delirium often self-resolve with time. • Nonpharmacologic interventions should be implemented to manage the delirium, such as ensuring the patient receives proper nutrition, hydration, comfort, and sleep and other situation-specific interventions.
Ophthalmologic condition	• Patient education about the condition should be provided. • Management depends on specific condition. • Referral to ophthalmologist is typically indicated. • For CBS, in some cases in which the vision impairment cannot be corrected and the visual hallucinations are distressing for the patient and impairing their quality of life, off-label use of certain medications, such as antipsychotics, antiepileptics, or SSRIs, may be considered. • For CBS, encourage the patient to engage in counseling or support groups.

(Continues)

Table 2. Treatments for the underlying causes of visual hallucinations *(continued)*

LBD	• Consultation with a neurologist or neurodegenerative disease specialist is advisable for a comprehensive treatment plan. • Nonpharmacologic interventions such as environmental modification should be implemented. • Cholinesterase inhibitors (for example, donepezil*) may be used to address cognitive symptoms. • Off-label use of antipsychotic medications may be considered for hallucinations but should be used with extreme caution as these medications have a boxed warning for increased mortality in older adults with dementia-related psychosis. Additionally, patients with LBD may have neuroleptic sensitivity, in which they experience negative reactions to use of antipsychotics, such as worsening of symptoms, severe parkinsonism, or mental status changes.
Medication adverse reaction	• Whenever possible, consideration should be given to discontinuing the offending medication, switching to an alternative medication, or decreasing the dose.

Abbreviations: CBS, Charles Bonnet syndrome; CGRP, calcitonin gene-related peptide; LBD, Lewy body dementia; NSAIDs, nonsteroidal anti-inflammatory drugs; PMHNP, psychiatric-mental health NP; SSRIs, selective serotonin reuptake inhibitors.

*Off-label for LBD.

Note: Table does not constitute a comprehensive overview of treatment. Providers should review appropriate guidelines and other resources for more information.

Alzheimer disease (AD).[20] The study also found that visual hallucinations were associated with limbic Lewy body pathology in LBD, whereas visual misperceptions and misidentification delusions were associated with the cortical Lewy body and neurofibrillary tangle burden in LBD and AD. To aid in diagnosis of the patient, a thorough subjective assessment, including details about the hallucinations such as their nature, content, and frequency, should be done. The patient should also be assessed for cognition, memory, and mood changes and for motor symptoms such as parkinsonism. Family history should be investigated, since there are strong genetic links to LBD. The patient's current medication regimen and any medication changes should be evaluated, as some medications can exacerbate visual hallucinations in LBD.

Medication adverse reactions

It is important for PCPs to be aware that certain medications may cause visual adverse reactions. A number of psychiatric medications, such as olanzapine, quetiapine, haloperidol, clonazepam, and lorazepam; the dopamine agonist ropinirole; sedative-hypnotic medications, including zolpidem and eszopiclone; and some antiepileptic medications, such as gabapentin and pregabalin, may cause visual hallucinations or worsen preexisting visual hallucinations. This list is inexhaustive; therefore, it is vital to review potential adverse reactions of any medications a patient is taking.

Selecting an appropriate intervention

Visual hallucinations have many causes. A thorough subjective and objective assessment helps to enable determination of the best course of treatment. *Table 2* outlines various treatments that may be considered for patients with the listed conditions.

Conclusion

Visual hallucinations can arise from a variety of causes, including psychiatric disorders, delirium, infectious diseases such as HIV and syphilis, metabolic disorders such as hyperammonemia, neurologic conditions such as LBD, ophthalmologic conditions such as CBS, and substance use, as well as other conditions not described in this article. Identifying the underlying cause is crucial for appropriate management and treatment. NPs, as integral members of the healthcare team, play a pivotal role in assessing and diagnosing patients with visual hallucinations. A thorough medical history and physical exam along with appropriate lab and imaging studies can aid in the differential diagnosis of visual hallucinations and guide appropriate management strategies. It is important to consider all possible causes for patient presentation with visual hallucinations to drive prompt management of the symptom. NP

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Pauline Stoltzner is an assistant professor at the Orvis School of Nursing at the University of Nevada, Reno in Reno, Nev. and chief executive officer and APRN at Advanced Practice Primary Care in Reno, Nev.

Cameron Duncan is interim dean of nursing and an associate professor at the Orvis School of Nursing at the University of Nevada, Reno in Reno, Nev. and chief executive officer and APRN at Duncan Family Healthcare in Reno, Nev.

The authors and planners have disclosed no potential conflicts of interest, financial or otherwise.

DOI-10.1097/01.NPR.0000000000000139