Parkinson's disease who possibly exhibits this rare form of perceptual disturbance.

Case Report

Our patient is a 76-year-old Caucasian woman with a 3-year history of Parkinson's disease, presenting with acute dyspnea which resulted in her hospitalization. She reported visual hallucinations of "mechanical bugs walking around the hospital" and "two people fighting" in the corner of her room. The visual hallucinations began 1 year prior to her hospitalization and were nondistressing. She had insight into the visual hallucinations. Our patient was treated with carbidopa-L-dopa until 4 months prior to admission. The patient's mental status exam was notable for bradyphasia, psychomotor retardation, and limited range of affect. She had signs of a pill rolling tremor on her left hand. She was otherwise alert and oriented times 3 with no fluctuations in consciousness. She reports visual hallucinations in the absence of bizarre delusions or auditory hallucinations. Cognitive examination was notable for deficits in short-term recall (which improved with cues) and attention span.

An MRI scan of the brain revealed a hyper-intense focus adjacent to the right thalamus consistent with an old lacunar infarct. An EEG revealed no eliptiform discharges or significant slowing.

Our patient was treated with quetiapine for the visual hallucinations, with a noted decrease in the number of hallucinations for the remainder of the hospital stay.

Discussion

Visual hallucinations often suggest a wide range of etiologies. Hallucinations and delusions occur in up to 40% of patients with Parkinson's disease. Visual hallucinations are typically associated as a side effect of dopamine agonists, such as carbidopa-L-dopa, in about 20% of Parkinson's disease patients; however, our patient's visual hallucinations persisted despite discontinuation of carbidopa-L-dopa.[2] The differential diagnosis for visual hallucinations includes neurodegenerative dementias, such as Parkinson's dementia, postictal states, intoxications/delirium tremens, migraine headache with aura, and narcolepsy.[3]

Peduncular hallucinosis is a rare form of visual hallucination characterized by intense, vividly colored, nonstereotypical visual images of people, animals, and plants that are nonthreatening to the patient. The exact mechanism for peduncular hallucinosis is unknown. One theory is that when "normal" afferent input is decreased, for example by diminished visual acuity, spontaneous cerebral activity of the visual system is disinhibited, resulting in visual hallucinations. However, given the rarity of such cases, it may be that cerebral pathology may render some elderly patients vulnerable to this disinhibited phenomena associated with pontine lesions.[4] However, according to Cubo et al.,[5] visual hallucinations are more likely to occur with more severe overall Parkinson's symptoms and longer duration of Parkinson's disease. Our patient only had Parkinson's disease for 3 years and was only mildly impaired by the illness. Thus, based on clinical and radiographic findings, peduncular hallucinosis was considered in the differential diagnosis of our patient's visual hallucinations. In closing, the emergence of new-onset visual hallucinations in the elderly warrants an MRI of the brain and, although rare, peduncular hallucinosis should be considered in the differential diagnosis, especially with brain stem or thalamic infarcts.

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The Role of Melatonin in Sleep Disturbances in End-Stage Huntington's Disease

To the Editor: Sleep disturbances are common in Huntington's disease and are often characterized by disruption of day-night patterns.[1–3] In general, these sleep disturbances are attributed to factors of comorbidity (depression, mania), medication, or specific symptoms such as chorea or dystonia.

Circadian sleep is regulated by the "biological clock" or "pacemaker" of the suprachiasmatic nucleus. This pacemaker stimulates melatonin synthesis in the pineal gland. Clock genes play a central role in this molecular oscillation process. In an animal model of Huntington's disease, a marked

disruption of expression of the clock genes *mPer2* and *mBal* was found in the suprachiasmatic nucleus.[3] This may have negative consequences for the production of melatonin and the circadian sleep rhythm.

We evaluated the occurrence of circadian sleep disorders in end-stage Huntington's disease by determining deviations of dim light melatonin onset out of the saliva of 10 Huntington's disease patients with a sleep disorder according to DSM-IV-TR criteria. Patients were all residents in a specialized Huntington's disease ward of a nursing home who required constant care because of the severity of their disease.

Dim light melatonin onset is the most accurate marker for assessing the circadian pacemaker. It is defined as the time at which a salivary concentration of 4 pg/ml is reached.[4] Normally, this concentration is reached in adults between 7:30 p.m. and 10:00 p.m.[5] Dim light melatonin onset was determined by obtaining hourly saliva samples (from 9:00 p.m. to 1:00 a.m.) by chewing on a cotton plug for 1 minute (Salivetten, Sarstedt Etten-Leur, the Netherlands). Because of the risk of choking the researcher used a plastic clip to hold the cotton plug in place in the patient's mouth. In the evening, patients were held to food restrictions and were requested to avoid physical strain and bright light. Normal medication use was continued to maintain therapeutic blood levels and to avoid disruption of normal routine.

Dim light melatonin onset was identified in five of the 10 patients. One patient reached the melatonin concentration of 4 pg/ml in saliva before 10:00 p.m. In four of the five patients, dim light melatonin onset was identified between 10:00 p.m. and 12:00 a.m. One patient did not reach the concentration of 4 pg/ml before 1:00 a.m. In one patient, melatonin could not be detected in saliva samples, and in another, the melatonin assessment failed due to an insufficient quantity of saliva. Two patients dropped out. When dim light melatonin onset data were combined with circadian sleep disturbances, we found two patients with delayed sleep symptoms with dim light melatonin onset between 10:00 p.m. and 12:00 a.m. One patient (with undetectable saliva melatonin) showed advanced sleep symptoms.

In conclusion, although sleep research is difficult in severe end-stage Huntington's disease, the results of our study did provide some support for the hypothesis that there is a relation between Huntington's disease and circadian sleep disturbances that could be caused by melatonin deficiency. Further research using an experimental treatment with melatonin in a placebo-controlled setting is recommended.

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Co-occurrence of X-Linked Congenital Adrenal Hypoplasia and Autistic Disorder

To the Editor: One form of congenital adrenal hypoplasia is associated with the Xp21 chromosomal region. These patients have clinical abnormalities including mental retardation, hearing loss, hypogonadism, glycerol kinase deficiency, ornithine transcarbamoylase deficiency, and Duchenne muscular dystrophy. This letter presents a case of a patient with X-linked congenital adrenal hypoplasia whose mental status as a preadolescent male was also consistent with the diagnosis of autistic disorder.

Case Report

A 12-year-old Caucasian boy presented with features of autistic disorder including marked lack of awareness of the feelings of others; no seeking of comfort in times of distress; gross impairment in ability to make peer friendships; abnormal nonverbal communication; abnormalities in speech pro-