

Peduncular Hallucinosi s: A Case Report

Lauren Penney, MBBS,¹ David Galarneau, MD^{1,2}

¹The University of Queensland School of Medicine, Ochsner Clinical School, New Orleans, LA

²Department of Psychiatry, Ochsner Clinic Foundation, New Orleans, LA

ABSTRACT

Background: Peduncular hallucinosi s is a rare form of visual hallucination often described as vivid, colorful visions of people and animals. The exact pathophysiology is unknown; however, most cases have been described in relation to lesions in the thalamus or midbrain.

Case Report: We present the case of a 59-year-old female with peduncular hallucinosi s associated with infarction in the right basal ganglia with the background of malignant hypertension. The patient's visual hallucinations decreased without pharmaceutical treatment by the time of discharge and on further follow-up had resolved completely.

Conclusion: We believe ours is one of few reported cases of peduncular hallucinosi s in a patient with an infarct isolated to the basal ganglia (striatum and globus pallidus).

INTRODUCTION

Peduncular hallucinosi s is a rare form of visual hallucination first described by Lhermitte in 1922. The visions are usually reported to be vivid, colorful,¹ and sometimes distorted images of animals and people. They are typically considered nonthreatening by the patient. Peduncular hallucinosi s has often been described in relation to both vascular and infective lesions of the mesencephalon and thalamus.

Cases of peduncular hallucinosi s have been reported involving infarction of the basal ganglia as well as the thalamus.² We report a case of peduncular

hallucinosi s secondary to infarction in the basal ganglia only. To the best of our knowledge, few cases involving only the basal ganglia have been reported.

CASE REPORT

A 59-year-old female presented to the hospital with a 3-week history of right-sided headache and a 1-week history of irritability, confusion, and visual hallucinations with a background of malignant hypertension and hyperlipidemia. One week prior to hospital admission, she had presented to an outpatient clinic because of the headaches and was diagnosed with malignant hypertension and sinusitis. Magnetic resonance imaging revealed an infarct in the right globus pallidus, putamen, caudate, and anterior limb of the internal capsule. Magnetic resonance angiography showed multifocal areas of significant stenosis. The patient was transferred to our institution for evaluation by vascular neurology. Psychiatry was consulted in regard to the visual hallucinations.

The patient described visions of animals and people that ranged from shadows to bright colors, as well as Lilliputian hallucinations (hallucinations of people and objects of reduced size). She found these hallucinations nonthreatening and was aware that they were not real. Visions occurred in daylight and at night and were not associated with sleep disturbance, agitation, or visual disorders. The patient had no past psychiatric history and denied auditory hallucinations and delusions. She was alert and orientated to person, place, and time. Cognition, attention, memory, and language were intact.

The patient's medical history included malignant hypertension with diastolic dysfunction and hyperlipidemia. She had been noncompliant with medication for the hypertension for about 3 years. Family history was significant for her father having heart disease and transient ischemic attacks in his 60s. The patient did not smoke or drink alcohol and was not currently taking any medication except an antibiotic prescribed 1 week prior for sinusitis. During admission, the patient was newly diagnosed with type 2 diabetes mellitus, with a hemoglobin A1c of 6.7%. She was

Address correspondence to
David Galarneau, MD
Department of Psychiatry
Ochsner Clinic Foundation
1514 Jefferson Hwy.
New Orleans, LA 70121
Tel: (504) 842-4025
Email: dgalarneau@ochsner.org

Keywords: Basal ganglia, hallucinations, infarction

The authors have no financial or proprietary interest in the subject matter of this article.

also found to have chronic kidney disease stage 3 with a baseline creatinine of 1.82 mg/dL.

General physical examination was unremarkable. On neurologic examination, cranial nerves II-XII were intact and equal bilaterally. Upper and lower extremity motor strength was 5/5, and sensation was normal. The patient was treated with clopidogrel bisulfate (Plavix) and aspirin for the infarct, metoprolol for hypertension, and atorvastatin calcium (Lipitor) for hyperlipidemia. Her hypertension was treated with a combination of metoprolol, nifedipine, hydralazine, isosorbide mononitrate, and a clonidine patch. Angiotensin-converting enzyme inhibitors were avoided because of the patient's decreased renal function.

Treatment of the hallucinations with an antipsychotic was discussed with the patient, but because of the medication's side effects and the fact that the patient was not bothered by the visual hallucinations, pharmaceutical treatment was not recommended. At the time of discharge, the visual hallucinations were still present but decreasing in frequency. At further follow-up, they had completely resolved.

DISCUSSION

Peduncular hallucinosis is characterized by visual hallucinations of concrete objects that are often vivid and colorful. The patient does not mistake these visions for reality, which is an important distinction between hallucinosis and psychiatric visual hallucination. This condition has primarily been reported in single case reports, so information about associated symptoms is inconsistent.³ The exact lesion and pathogenesis of peduncular hallucinosis are still unknown, although many cases involve vascular lesions in the midbrain or thalamus.

Peduncular hallucinosis has commonly been described in the setting of sleep-wake cycle disturbance, with visions more pronounced nocturnally.¹ This association led Lhermitte to propose that sleep disturbance was the decisive factor in the condition.¹ However, although sleep disturbance is common in peduncular hallucinosis, it is not essential.² In our case, the patient experienced visual hallucinations day and night and did not exhibit any sleep cycle dysfunction.

Vascular lesions have been reported as the most common cause of peduncular hallucinosis, with the thalamus, midbrain, and brainstem most commonly affected.⁴ Similar to many reported cases, our patient developed peduncular hallucinosis as a result of an infarction. However, our patient's infarct was limited to the right basal ganglia only with no direct involvement of the thalamus or midbrain.

Determining how visual hallucinations can result from lesions in areas of the brain that are not part of the visual pathway has been difficult. Two common mechanisms have been suggested: imbalance between neurotransmitters in the reticular activating system (RAS),² and disruption of the basal ganglia (temporal lobe loop).⁵

The RAS may be involved in the production of peduncular hallucinosis because of the association with sleep-wake cycle disturbance. The RAS is composed of neuronal circuits connecting the brainstem to the cortex and is responsible for regulating arousal and sleep-wake cycles. Lesions are thought to alter the ponto-geniculo-occipital (PGO) waves that are associated with rapid eye movement (REM) sleep. Interruption to serotonergic inhibitory afferents in the dorsal raphe nuclei are suspended, resulting in an increase of PGO waves and thus an increase in REM sleep. Hallucinations may result from patients entering REM sleep quickly from a higher level of arousal.²

Another possible mechanism for peduncular hallucinosis involves a closed loop between the basal ganglia and the inferotemporal lobe.⁵ The basal ganglia are primarily known to control movement and coordination, but Middleton and Strick suggested that the inferotemporal lobe, which is responsible for recognition and discrimination of visual objects, may also be an output target for the basal ganglia.⁵ The basal ganglia loop involves a direct pathway (through the substantia nigra pars reticulata and internal globus pallidus complex) and an indirect pathway (through the external globus pallidus and subthalamic nucleus) to the temporal lobe via the thalamus. Middleton and Strick hypothesized that lesions of the substantia nigra and brainstem compression may result in visual hallucinations by blocking the stimulatory signal from the subthalamic nucleus to the substantia nigra, in turn decreasing the inhibitory signal to the thalamus and resulting in overactivity of the thalamus and the inferotemporal lobe. This mechanism may be responsible for the peduncular hallucinosis experienced by our patient after suffering an infarction in the striatum and globus pallidus.

In most situations, as in our case, peduncular hallucinosis is self-limiting and does not require any treatment. One study suggested that the atypical antipsychotic olanzapine could be of potential benefit.⁶ However, in the majority of cases reviewed, the visual hallucinations experienced by the patient resolved on their own.

CONCLUSION

Peduncular hallucinosis is a rare form of visual hallucinations most commonly caused by lesions to

the midbrain and thalamus, either alone or in combination with other areas of the brain. It may also present in the setting of other neurological disease, such as multiple sclerosis, or as the result of medications. We have presented what we believe to be one of few reported cases of peduncular hallucinosis in a patient with an infarct isolated to the basal ganglia (striatum and globus pallidus).

ACKNOWLEDGMENTS

The authors would like to thank the following for their advice and care of this patient: Dr Michael Knight, Department of Psychiatry, Ochsner Medical Center; Dr Erin Capone, Department of Psychiatry, Ochsner Medical Center; and Dr Draga Jichici, Department of Neurology, McMaster University.

REFERENCES

1. Lhermitte J. Syndrome de la calotte du pedoncule cerebral. les troubles psycho-sensoriels dans les lesions du mesocephale. *Rev Neurol.* 1922;38:1359-1365.
2. Manfred M, Andermann F. Complex visual hallucinations. Clinical and neurobiological insights. *Brain.* 1998 Oct;121(Pt 10):1819-1840.
3. Benke T. Peduncular hallucinosis: a syndrome of impaired reality monitoring. *J Neurol.* 2006 Dec;253(12):1561-1571. Epub 2006 Sep 27.
4. Kölmel HW. Peduncular hallucinations. *J Neurol.* 1991 Dec;238(8):457-459.
5. Middleton FA, Strick PL. The temporal lobe is a target of output from the basal ganglia. *Proc Natl Acad Sci U S A.* 1996 Aug 6; 93(16):8683-8687.
6. Spiegel D, Barber J, Somova M. A potential case of peduncular hallucinosis treated successfully with olanzapine. *Clin Schizophr Relat Psychoses.* 2011 Apr;5(1):50-53.

This article meets the Accreditation Council for Graduate Medical Education and the American Board of Medical Specialties Maintenance of Certification competencies for Patient Care and Medical Knowledge.