

A Potential Case of Peduncular Hallucinosi Treated Successfully with Olanzapine

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Abstract

Visual hallucinations have a differential diagnosis, both psychiatric and nonpsychiatric in nature. Described first by Lhermitte, peduncular hallucinosis is an uncommon etiology of visual hallucinations (VH). Typically, the offending lesion is vascular in origin and occurs at the level of the midbrain, thalamus, or rostral brainstem. Interestingly, the origin of the VH in our patient's case could have been either/both from an ischemic insult at the midbrain or compression of the brainstem due to aneurism. While evidence for treatment is scarce, we present a posited case of peduncular hallucinosis treated successfully with olanzapine.

Key Words: Psychoses/Visual Hallucinations, Neurology, Magnetic Resonance Imaging, Olanzapine

Introduction

Visual hallucinations (VH) have a broad differential diagnosis including peduncular hallucinosis (PH) (1). PH, first described by Lhermitte, included symptoms of VH with nocturnal insomnia and daytime somnolence (2). The hallucinations are usually well-formed, with vivid colors and detailed images (1). Lilliputian hallucinations, in which the figures or objects are shrunken but still fully detailed, are a common aspect of PH. Patients usually, but not always, retain insight (1).

Although there are several theories speculating on the cause of PH, there is presently no one accepted etiology. One

theory is that the brainstem plays an integral role in suppressing visual hallucinations, and the spontaneous activity of the visual system increases if this is disrupted, leading to hallucinations (3, 4).

The following case illustrates a possible case of PH successfully ameliorated with olanzapine (OLZ).

Case Report

Our patient was a 77-year-old female who presented to the Emergency Room with sudden onset intermittent headache, diplopia and right eye ptosis, and an episode of visual hallucinations. On presentation, the patient was alert and oriented x3, and her husband denied any mental status change. Our patient did not have any metabolic disturbances, including hypo/hyper-natremia or kalemia, or renal/hepatic disease. Additionally, she had no endocrinopathies, vitamin deficiency states, inflammatory or infectious diseases. A magnetic resonance angiography revealed a 5x5 mm irregular right posterior communicating artery aneurysm with no evidence of hemorrhage, while

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magnetic resonance imaging demonstrated multiple foci of T2 weighted high signal intensity throughout the periventricular white matter and an acute/subacute right midbrain infarct. Neurosurgery believed that the third nerve palsy was caused by an expansion of the aneurysm, thus the presumed need for surgical intervention. The patient's family elected to proceed with an uneventful surgical artery clipping.

The patient's past medical history was significant for hyperlipidemia, treated with simvastatin. The patient denied alcohol or illicit substances usage.

On post-operative day (POD) 2, the patient's mental status changed, and she became "confused." Our patient developed VH, reporting seeing "little people" walking around the room. We were consulted on POD 5. On examination, the patient was awake, but with painful, right eye ptosis, and dilated right pupil, which was not responsive to light. The patient seemed to have minimal insight into the VH. Cognitively, despite being oriented only to person and with short-term memory impairment, she was able to complete the Vigilant A section of the Confusion Assessment Method (5) and scored zero on the Richmond Agitation Sedation Scale (6). This was in contrast to the previous four days, when she was described as having more fluctuation in her alertness. Her Folstein Mini-Mental State Examination score was 23/30 (7).

Other than the aforementioned ophthalmological findings, the remainder of our patient's physical and neurological examination was within normal limits. There was no reported family history of psychiatric illness or previous personal episodes of VH, save immediately prior to admission.

An electroencephalography showed no seizure activity, although it did reveal slowing to 7 Hz. The metabolic work-up was within normal limits, and chest X-ray and urinalysis were negative for infection. Our patient began OLZ 5 mg BID.

Three days after starting OLZ, the patient/family reported that she was no longer having VH, although the other mental status changes were still present. The patient went four days without any hallucinations, but then recurred, albeit with much less frequency (~q two days). After two weeks of OLZ therapy, the patient/family reported no hallucinations, and OLZ was discontinued. Unfortunately, the patient was lost to follow-up.

Discussion

Two of the most common features of PH are VH and sleep disturbances. The abrupt onset of VH along with confusion occurs in PH due to the vascular etiology (3). Most reported cases are due to a vascular insult to some portion of the rostral brainstem or thalamus (such as infarction, vasospasm, or compression).

Sleep disturbances in PH have not been thoroughly explored to date, but involvement of the ascending reticular activating system (ARAS) has been posited. The ARAS spans the brainstem, eventually giving off projections to the thalamus. It is located deep in the brainstem and is essential to consciousness and waking. Disruption of the blood supply due to the posterior communicating artery (PCoA) aneurysm, as occurred in our patient, could affect both the pons and the thalamus, leading to sleep disturbances attributed to the disruption of ARAS impulses from the brainstem reticular formation (6). Alternatively, expansion of the aneurysm itself could impinge upon the brainstem, causing compression, resulting in similar effects to the ARAS (17). Our patient had a lesion in the brainstem, more specifically in the right midbrain, which could explain her VH.

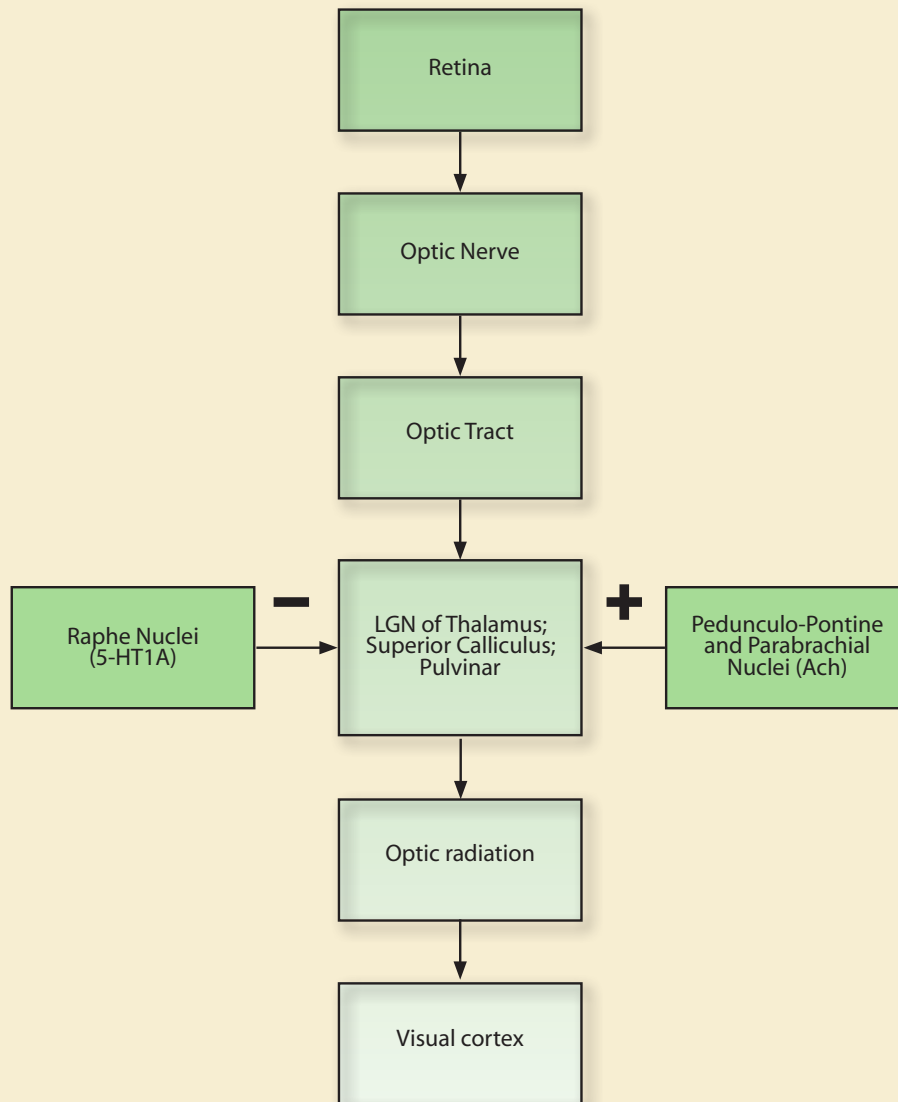
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While the neurobiology of visual hallucinations (VH) is still uncertain, there are certain concepts that may be stated, based on the anatomy of the retinogeniculocalacrine (RGC) tract and physiology of visual pathway transmission as follows: visual stimuli → retina → optic nerve → optic chiasm/tracts → lateral geniculate nucleus (LGN) of the thalamus. The LGN also receives input from the superior colliculus via the pulvinar, and then projects to the optic radiation through the temporal lobe into the visual cortex. Additionally, LGN transmission is modulated from the brainstem via excitatory cholinergic centers (pedunculo-pontine and parabrachial nuclei) and inhibitory serotonergic centers (dorsal raphe) (see Figure 1). It has been proposed that brainstem lesions involving the dorsal raphe system can result in loss of ascending serotonergic inhibition to the dorsal LGN, which may lead to an unopposed ascending cholinergic excitability to the LGN (10).

We suggest that the dual action of OLZ as a dopamine 2/serotonin 2A receptor antagonist (11) could have an important role in the treatment of PH. OLZ also has been shown to improve sleep continuity, sleep quality, and increase slow wave sleep (12), a potentially unforeseen benefit in PH because sleep disturbances are typically present.

One potentially unique aspect about our patient was the right oculomotor nerve palsy with right papillary dilatation, ptosis, and lack of reactivity to light. Because the most common lesion compressing the oculomotor nerve in the

Figure 1 Schematic of Visual Pathway Transmission



subarachnoid space is an aneurysm (13), and our patient did have a right posterior communicating artery aneurysm, we could have expected right papillary dilatation followed by other signs of third cranial nerve dysfunction, including ophthalmoparesis and ptosis.

The surprising finding of the midbrain infarct potentially provided us with two possible etiologies for PH, including the midbrain infarct itself or perhaps compression of the rostral portion of the brainstem by the PCoA aneurysm, which was the probable insult to the right third cranial nerve palsy. There are reports of detection of an aneurysm compressing the nerve in the setting of nontraumatic isolated third cranial nerve palsy (ITNP). In most patients, the

causative aneurysm is located at the junction of the internal carotid and posterior communicating arteries, at the apex of the basilar artery, or at its junction with the posterior cerebral or superior cerebellar arteries, and usually measures ≥ 4 mm in cross section (18).

It is unclear as to the acute development of a third nerve palsy and PH, although an undetected intraoperative complication, such as the rupture of the PCOM aneurysm, might explain this. For instance, there is a report of PH being the sole neurological manifestation after the rupture of an aneurysm, with cerebral vasospasm in the perforating arteries of the ascending reticular activating system defined as the probable cause of the hallucinosis (8). Additionally, pedun-

cular hallucinosis might be a manifestation of delayed cerebral ischemia after a subarachnoid hemorrhage (19).

There are some limitations to this case report. Firstly, PH may have spontaneous improvement within days of the initial onset (1), so it is not certain that OLZ caused the cessation of symptoms. Secondly, it is also possible that our patient's VH were caused by delirium post-operatively (14) or, plausibly, an underlying dementia (15). With respect to the latter, the acute onset of VH seems to lessen, but not eliminate, the likelihood of being due solely to dementia (15). Additionally, while the patient's symptoms of delirium (post-operatively) seemed to have significantly abated by our evaluation, it cannot be affirmed that her visual hallucinations were still not due to encephalopathy. A final limitation to our paper is the fact that we were not able to perform post-operative follow-up imaging; therefore, we couldn't fully assess the size of the aneurysm that was accountable for the compression. Despite these limitations, we feel that our proposal of PH is the most likely etiology of her VH.

Evidence for pharmacological management of PH is limited; however, there are reports of successful treatment of PH with anticonvulsants (16), and serotonergic-based therapies (9), as well as other atypical antipsychotics (17). Although not supported in all studies (10), but supported by our case report with the improvement seen in our patient after administration of OLZ, we suggest that atypical antipsychotics may have a role in the treatment of PH.

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