wjpmr, 2020,6(3), 151-155

SJIF Impact Factor: 5.922

WORLD JOURNAL OF PHARMACEUTICAL AND MEDICAL RESEARCH

www.wjpmr.com

Case Report

ISSN 2455-3301

WJPMR

A CASE REPORT OF PEDUNCULAR HALLUCINOSIS SECONDARY TO BRAIN STEM LESION

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Article Received on 25/12/2019 **Article Revised on 15/01/2020** **Article Accepted on 05/02/2020**

ABSTRACT

Peduncular hallucinosis (PH) is a rare complex visual hallucinations often presented as vivid, colorful visions of people, animals and objects. The exact etiology of peduncular hallucinosis is not known but several hypothetical mechanisms were explained by different clinicians. We presented a case of 65-year-old female with visual hallucinations with brain stem lesion in the form of hypertrophic olivary degeneration with the background of diabetes.

KEYWORDS: Peduncular Hallucinosis, Brain stem lesion, Hypertrophic olivary degeneration, Diabetes.

INTRODUCTION

Peduncular hallucinosis (PH) is a very rare form of complex visual hallucination first described by Lhermitte in the year of 1922.[1] The hallucinations are usually well-formed and explained to be vivid colors and detailed images and sometimes distorted images of animals, people and objects.[2] The common aspect of PH is Lilliputian hallucinations, in which images or things are seen to be small in size but visible clearly. Usually the patients with PH are not always retaininsight; mostly insight is present in majority of the cases. Exact etiology of PH was idiopathic but several theories stating that vascular lesions or infectious lesions of the midbrain, pons, thalamus.[3] were the cause and in many of the cases of PH involve infarction basal ganglia and thalamus and few cases reported that ocular degeneration, cataract cause PH. Several pathological theories are explained but most accepted among them are disturbances in RAS (Reticular activating system), injury to dorsal raphe nuclei and closed loop between basal ganglia and inferotemporal lobe.[4] Diagnosis of PH was major challenge to the physicians because of its complexity and differential diagnosis for psychiatric visual hallucinations and for visual hallucinations in narcolepsy-cataplexy syndrome, parkinson's disease, lewy body dementia etc.

CASE REPORT

A 65-year-old female presented to us with complaints of reduced vision in both eyes of 9 months duration and visual hallucinations of 2 months duration. Visual disturbances complained are gradually progressive painless vision loss in both eyes of 9 months duration. For these complaints they have consulted ophthalmologist and underwent cataract removal in right eye but vision loss did not improve and progressed instead.

For the past 3 months she is having visual hallucinations. Her description was that she sees a man who continuously plays damarukam (a small percussion musical instrument). He keeps poking at her, runs around her bed and calls a group of people and is instructing them something about the patient .Patient couldn't hear what he is telling and assumes that he is instructing them to kill her. He and the group of people accompany her to wash room and they sit outside and keep discussing which she assumes is about her. Visual hallucinations occurred in day light and at night and were associated with sleep disturbances.

There is no history of hallucinations in other sensory modalities, no h/o any symptoms of mania, psychosis or any other psychiatric disorders, memory or behavioural

disturbances, and no history any other focal neurologic deficits.

She has past h/o of pulmonay tuberculosis and has been started on ATT 5 months prior to onset of her vision loss. She is known diabetic and had bilateral renal stones for which she underwent ureteric stenting. Family history is nil significant.

On examination, her general condition is normal and vital data were within normal limits. There is no perception of light in her right eye, while it is intact in left eye. On fundoscopic examination, both optic discs were pale, with arteriolar narrowing. Left pupil is normal in size and reacting to light and right pupil operated. Other cranial nerve, motor sensory, cerebellar and basal ganglia and higher mental examination were found to be normal.

Patient has been, referred to psychiatric department, their impression was late onset schizophrenia and prescribed Risperidone 2mg once a day. Patient later referred to ophthalmology department for visual abnormalities, ophthalmologist after examination of her eyes they diagnosed that patient had bilateral optic atrophy.

Her routine blood investigations were found to be in normal limits except RBS-219mg%, and HbA1C-8.2%.

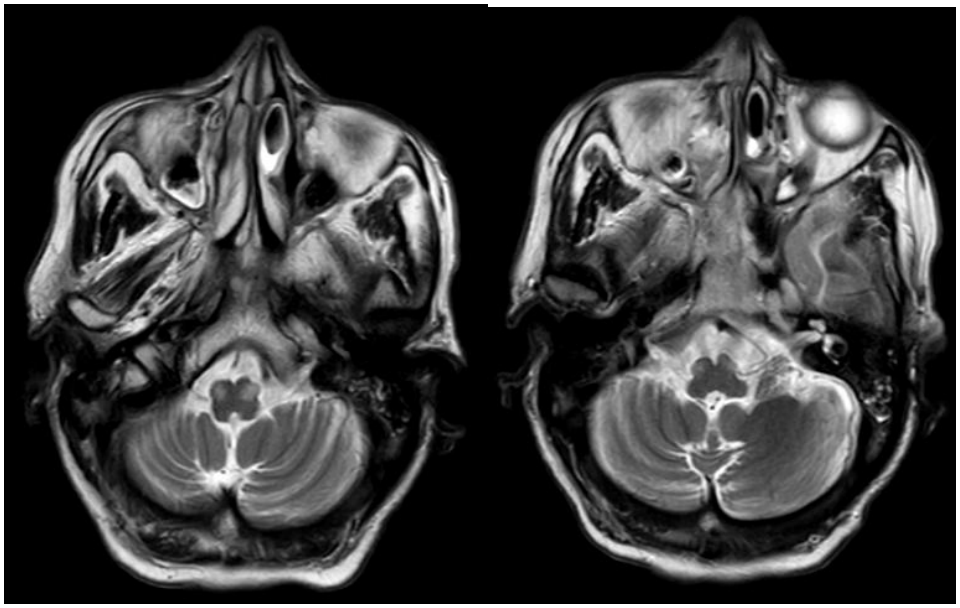


Figure 1: MRI T2 images.

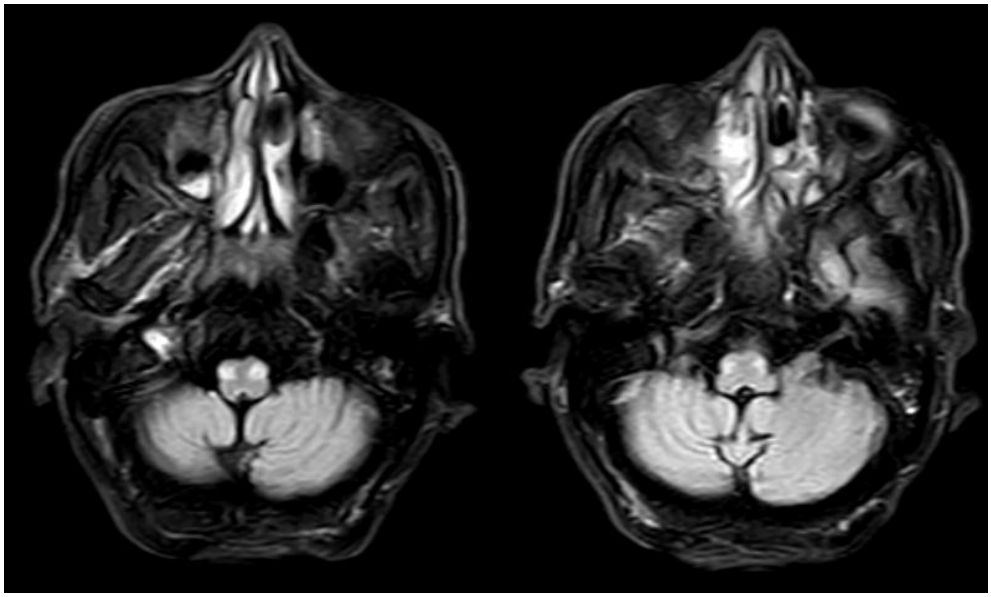
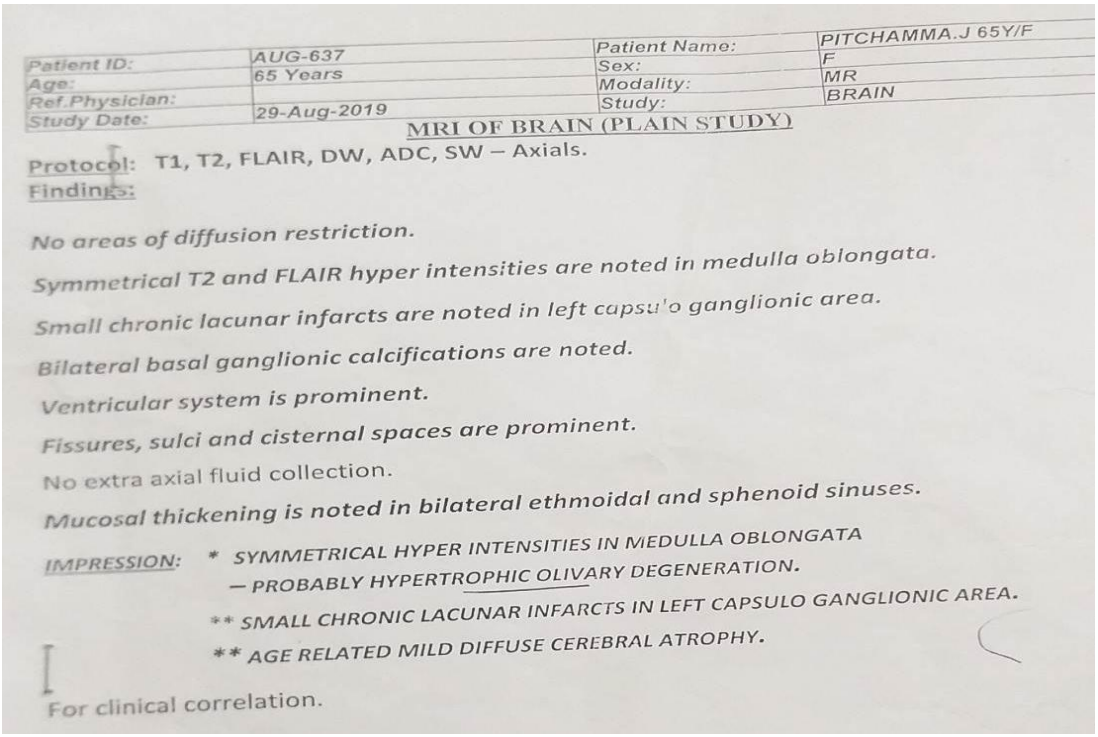


Figure 2: MRI flair images.



Patient ID:	AUG-637	Patient Name:	PITCHAMMA.J 65Y/F
Age:	65 Years	Sex:	F
Ref.Physician:		Modality:	MR
Study Date:	29-Aug-2019	Study:	BRAIN

MRI OF BRAIN (PLAIN STUDY)

Protocol: T1, T2, FLAIR, DW, ADC, SW – Axials.

Findings:

No areas of diffusion restriction.

Symmetrical T2 and FLAIR hyper intensities are noted in medulla oblongata.

Small chronic lacunar infarcts are noted in left capsulo ganglionic area.

Bilateral basal ganglionic calcifications are noted.

Ventricular system is prominent.

Fissures, sulci and cisternal spaces are prominent.

No extra axial fluid collection.

Mucosal thickening is noted in bilateral ethmoidal and sphenoid sinuses.

IMPRESSION: * SYMMETRICAL HYPER INTENSITIES IN MEDULLA OBLONGATA
— PROBABLY HYPERTROPHIC OLIVARY DEGENERATION.

** SMALL CHRONIC LACUNAR INFARCTS IN LEFT CAPSULO GANGLIONIC AREA.

** AGE RELATED MILD DIFFUSE CEREBRAL ATROPHY.

For clinical correlation.

Figure 3: MRI Findings.

With a history of visual hallucinations in the absence of auditory hallucinations and other behavioural disturbances, our first differential diagnosis was peduncular hallucinosis. With the imaging finding of a brain stem lesion, we attributed these hallucinations to disruption of ARAS. We have attributed vision loss to ethambutol toxicity but haven't considered vision loss as a cause for visual hallucinations because there is perception of light which means that there is some kind of input through the visual system.

DISCUSSION

Lhermitte in 1922, first described peduncular hallucinosis in a 72-years-old woman who initially developed the signs of stroke involving the cerebral peduncle and pons, she complained about 2 weeks of head ache, vomiting and diplopia. With these clinical and imaging findings, patient diagnosed with peduncular hallucinosis with mesencephalon lesion.[1] Later on second case reported in 1927, Van Bogaert presented the a similar case of a 59-year-old woman who, at autopsy, had findings of a stroke involving the pulvinar nucleus of the thalamus, his patient experienced peduncular hallucinosis due to degeneration of the periaqueductal gray, particularly the median raphe and the oculomotor nuclei.[5] Several cases were reported due to different vascular lesions in the thalamus and mid brain; these Complex visual hallucinations may be seen in a variety of disorders and must be considered at the differential diagnosis of PH. These consist of narcolepsy‑cataplexy syndrome, Parkinson's disease, Lewy body dementia, delirium, migraine coma, Charles Bonnet syndrome, Schizophrenia, hallucinogen induced states and epilepsy.[4]

The exact pathophysiology of peduncular hallucinosis is not known, but several mechanisms were posited by different clinicians. Manford and Andermann reported that peduncular hallucinosis results from disruption of visual processing pathways and the imbalance between neurotransmitters in the ascending reticular activating system (RAS) and also the disruption of the basal ganglion (temporal lobe loop).[4] RAS is a set of interconnected nuclei connecting the brainstem to the cortex and involved in regulating arousal and consciousness. In our case patient complained about sleep disturbance these maybe due to her RAS pathway disruption.

The neuropathology of visual hallucinations (VH) is still perplexed; 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 (fig-4).

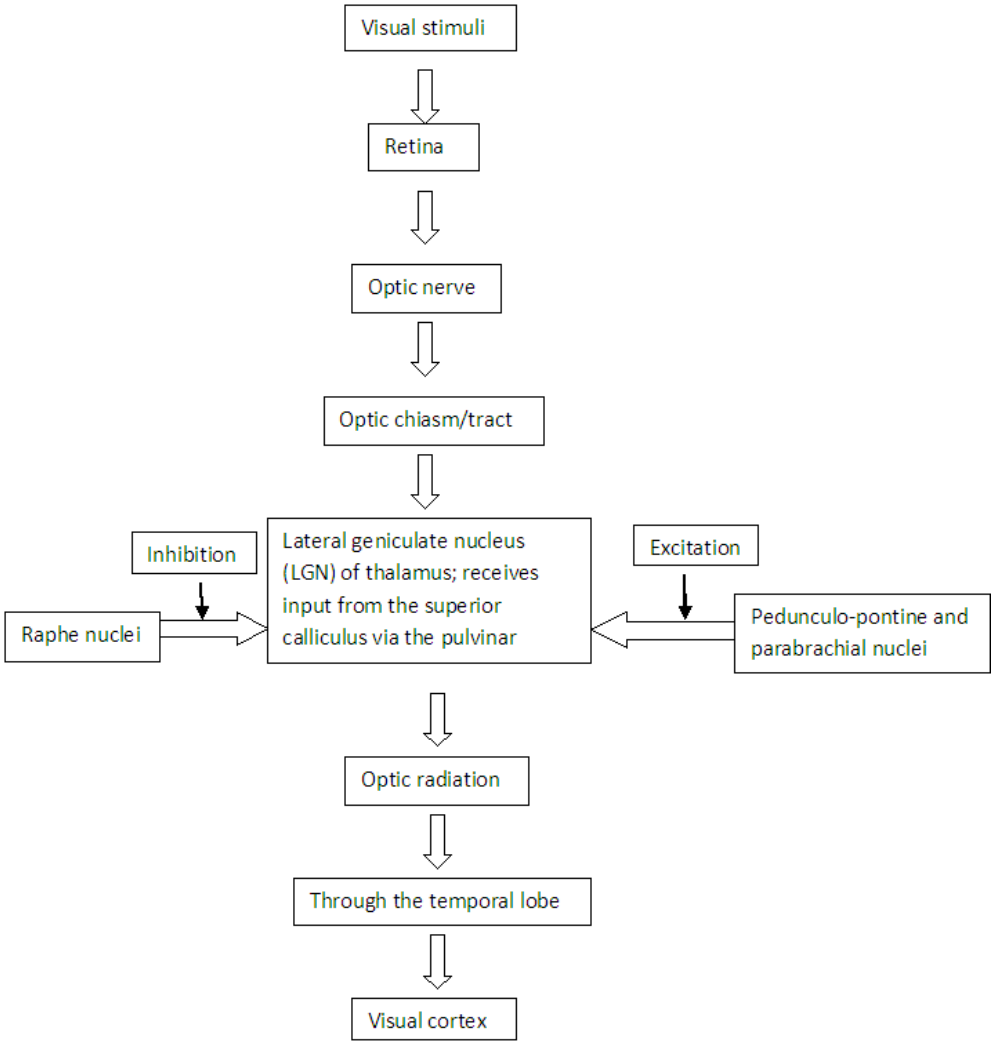


Figure 4: Visual pathway transmission.

LGN transmission is modulated from the brainstem via excitatory cholinergic centers (pedunculo-pontine and parabrachial nuclei) and inhibitory serotonergic centers (dorsal raphe) (Fig 4). It has been concluded 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 [6]. Specifically, injury to the dorsal raphe nuclei has the effect of impaired suppression of the dorsal lateral geniculate nucleus of the thalamus this may leads to complex visual hallucinations.

Peduncular hallucinosis is often caused by ischemic or hemorrhagic stroke injury to the rostral brainstem, it may also be the presenting symptom of several tumors like craniopharyngiomas, medulloblastomas, cerebellar juvenile pilocytic astrocytomas, pineal meningiomas, metastatic disease[7], pontine cavernomas, and posterior fossa meningiomas [8].The cause of the hallucinations in these cases is generally due to compression of the pons, midbrain, and/or diencephalon, and they improve after tumor suppression or resection. Another possible cause for this peduncular hallucinosis is aneurysmal subarachnoid hemorrhage due hypertensive crisis, stating that peduncular hallucinosis may occur as a result of brainstem ischemia from vasospasm.[9,10]

Another possible mechanism for peduncular hallucinosis involves due to close basal ganglia and inferotemporal lobe interconnecting loop. 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 globuspallidus and subthalamic nucleus) to the temporal lobe via the thalamus. Middleton and Strick[11] 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 over activity of the thalamus and the inferotemporal lobe. This mechanism was not responsible for PH of our patient because she did not have any infarct in the substantia nigra and in subthalamic nucleus.

Some studies showed that vascular lesion have been reported as the most common cause of peduncular

hallucinosis; with the thalamus, midbrain, and brainstem are the most commonly affected areas. Ocular pathology like macular degeneration, cataract were also be the reasons for visual hallucinations. There is no particular treatment for peduncular hallucinations, but some studies showed that antipsychotics may decrease the hallucinations.[12]

CONCLUSION

Peduncular hallucinosis(PH) is the rare form of complex visual hallucinations, the exact etiology of PH was not know several mechanism were explained but most commonly caused by lesions to the midbrain, thalamus and basal ganglion. Some studies showed that any degeneration to visual tracts in visual pathway, disruption of RAS system, ocular degenerations, cataract, tumors, aneurysms and for other neurological disorders these hallucinations were the presenting symptom. There is no particular treatment available to treat PH in most of the cases hallucinations were self-reducible, some cases required anti-psychotic drugs intervention. We reported the case of peduncular hallucinosis with brain stem lesion in the form of hypertrophic olivary degeneration with the medical history of diabetes.

ACKNOWLEDGEMENT

All the authors of this article like to take the privilege of expressing cordial thanks to management of Chalapathi Institute of Pharmaceutical Sciences and Government General Hospital, Guntur and also convey sincere gratitude for enabling to prepare this article under the close supervision of HOD of department of Neurology.

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