



Peduncular Hallucinosi due to Multiple Sclerosis: A Case Report

Multipl Skleroza Baęlı Pedinküler Halüsinoz: Olgu Sunumu

Özden Kamışlı, Yüksel Kaplan, Suat Kamışlı, Cemal Özcan
İnönü University Faculty of Medicine, Turgut Özal Medical Center, Department of Neurology, Malatya, Turkey

Summary

Peduncular hallucinosi is a syndrome characterized by vivid, colored visual hallucinations due to brain stem and thalamic involvement. The etiology of peduncular hallucinosi mostly involves lesions of vascular nature and infections. The cases of peduncular hallucinosi due to multiple sclerosis is very rare in the literature. In this report we presented a peduncular hallucinosi case who is a 46 year old woman with multiple sclerosis diagnosis. (*Turkish Journal of Neurology* 2013; 19:143-144)

Key Words: Peduncular hallucinosi, multiple sclerosis, brain stem

Özet

Pedinküler halüsinoz, beyin sapı ve talamus gibi yapıların etkilenmesiyle ortaya çıkan kısa süreli renkli, canlı görsel halüsinasyonlarla karakterize bir sendromdur. Etiyolojide daha çok vasküler hadiseler ve enfeksiyonlar yer almaktadır. Literatürde multipl skleroza baęlı pedinküler halüsinozis olgusu oldukça az sayıdadır. Biz bu yazıda multipl skleroz tanısı olan, 46 yaşındaki bir hastada gelişen pedinküler halüsinozisi sunduk. (*Türk Nöroloji Dergisi* 2013; 19:143-144)

Anahtar Kelimeler: Pedinküler halüsinozis, multipl skleroz, beyin sapı

Introduction

First described by Lhermitte, peduncular hallucinosi (PH) is a syndrome characterized by brief, colorful visual hallucinations (1, 2). It is often caused by involvement of mesencephalon. In addition, there have been reports mentioning vascular or infectious disorders of thalamus, substantia nigra-pars reticulata, pons and basal diencephalon as potential causes (2,3). Cases where PH emerges following a vertebral artery angiography or cardiac catheterization were also shown (1,3). In the literature, PH due to multiple sclerosis (MS) has been reported only once in the past (4). Here we present a second such case on the basis of the condition's rarity in the PH literature.

Case

The forty six year old patient previously diagnosed with relapsing-remitting MS has been visiting our clinic since 2001

and reported having daily hallucinations of long, yellow hair on her fingers, insects on her hair or people wearing bags on their head for 4-5 minutes almost every day for the past year. Her MS complaints started 20 years ago with difficulty in walking and urinary incontinence. She did not receive treatment for these symptoms and they went away in a month on their own. After two years, however, she had balance problems and blurry vision on the right eye. Again, she did not seek medical help at that time and the symptoms got better in time. She had some attacks with loss of balance and gait problems and, from time to time, some sensory complaints, but she was neither diagnosed nor sought treatment until 2001. After 2001, she received pulse steroid treatment for 5-7 days and interferon beta 1a as protective treatment. With the increased attack rate even when she was on interferon beta 1a, she was switched to glatiramer acetate in 2008. Her medical history did not show any other condition besides MS. She had been using glatiramer acetate for protective treatment since that time. Her familial history did not show an unusual condition.

Address for Correspondence/Yazışma Adresi: Özden Kamışlı MD, İnönü University Faculty of Medicine, Turgut Özal Medical Center, Department of Neurology, Malatya, Turkey Phone: +90 422 341 06 60-4905 E-mail: ozden.kamisli@inonu.edu.tr

Received/Geliş Tarihi: 07.01.2013 **Accepted/Kabul Tarihi:** 28.06.2013

The patient's physical examination was normal. In the neurological examination, right hemiparesis and ataxia as sequelae of MS. She scored 4.5 on the Expanded Disability Status Scale (EDSS). Complete blood count, biochemical and serological evaluations were all within normal ranges. Her electroencephalography (EEG) was normal. Brain magnetic resonance imaging (MRI) showed multiple demyelinated lesions in periventricular, left thalamus, pons left lateral section and left cerebral peduncle (Figure 1a-b). There were chronic stage T2 hyperintense lesions in cervical MR on C2-3, C3-4 and C7-T1 levels. For evoked potentials, there was an increased latency in P100 in visual evoked potentials. Somatosensory and brainstem auditory evoked potentials were within normal ranges. Oligoclonal band was seen to be positive in a previous cerebrospinal fluid test. While the presence of any active plaques in the early stage was unknown since she did not come in for an examination, there were no currently active contrast-defined lesions in her cranial MR. Her psychiatric evaluation did not suggest a primary psychopathology that would explain the hallucinations. Even though she reported seeing vivid and colorful hallucinations, she was able to reason that these were not real. Considering the brainstem and thalamus demyelination lesions, this condition was evaluated as PH. She was first started on Risperidone but she did not report a significant improvement on her symptoms. Later on, she was switched to Quetiapine 100 mg/day and her symptoms improved markedly.

Discussion

Lhermitte first described peduncular hallucinosis in a patient with mesencephalon lesion in 1922. Van Bogaert later made the pathological confirmation of the lesion location in 1927 (1,3). After this, many PH cases due to vascular or infectious disorders of mesencephalon were reported (2,5). A survey of neuroimaging studies showed that the involvements of reticular formation, medial meniscus, spinothalamic pathway, raphe nucleus and periaqueductal gray matter, 3rd, 4th 6th and 7th cranial nerve nuclei, medial longitudinal fasciculus, substantia nigra, nucleus ruber, tegmentum, cerebral peduncle, pons, paramedian thalamic region and pulvinar may cause PH (2).

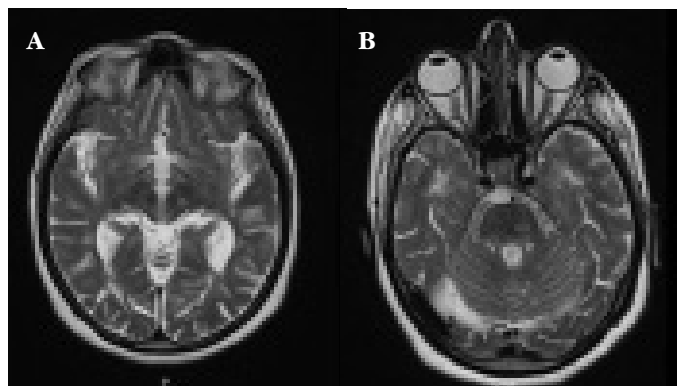


Figure 1. a) Left cerebral peduncle in T2 axial b) Left pons region hyperintense lesions

The source of lesions in PH is commonly of vascular nature. Less frequently, cases with brainstem or cerebellar tumors or infections were reported (2,5,6).

While the localization power of the visual hallucinations are not very strong, it is noteworthy that those seen in PH are often accompanied by symptoms indicating dysfunction of midbrain structures. Sleep disorders are often seen in patients with PH which suggests a loss of function in the ascendant reticular activating system (1). Attention deficit, reduction in cognitive agility, memory, executive functions and intellectual capacity are among other symptoms that can be seen in PH (2).

Two hypotheses have been proposed in the formation of PH. The first one is the imbalance between cholinergic, serotonergic and other neurotransmitters in the brainstem, leading to a dysfunction in the control mechanism regulating the input from brainstem to thalamus. This dysfunction may disrupt the filtering mechanisms in the other sensory cortices by modulating thalamic gating and cause hallucinations (2,7). The second hypothesis relates to the importance of the temporal lobe in the syndrome. In the event of disruption somewhere in the signal chain between substantia nigra, neostriatum, thalamus and visual cortex, there is an unusual input overflow from thalamus to visually responsive parts of the temporal lobe that could explain hallucinations (2,8). It has been seen that the lesions affecting raphe's dorsal nucleus temporarily cause an increase in ponto-geniculo-occipital spindles in sleep EEG, potentially leading to dreams which can be named as hallucinosis. It has been suggested that PH emergence due to a loss of inhibitory control in the ponto-geniculo-occipital system (6).

In our case, demyelination lesions in the brain stem and thalamus have caused PH. The fact that these hallucinations have been going on for a while and the lack of contrast-defined lesions in the cranial MRI suggested that this condition was not due to an acute attack. Our conclusion is that lesions of pons and cerebral peduncle affected ponto-geniculo-occipital pathways, reducing the inhibitory control and therefore caused hallucinations. This scenario may seem as a purely psychiatric case at first but it emphasizes the importance of careful study of possible organic causes in diagnosis and treatment. Our case was found to be worthy of a thorough report since it encourages a wider perspective in diagnostic approaches for patients with visual hallucinations.

References

1. Kamalakannan D, Ravi S, Moudgil SS. Peduncular hallucinosis: unusual complication of cardiac catheterization. *South Med J* 2004;97:999-1000.
2. Benke T. Peduncular hallucinosis: a syndrome of impaired reality monitoring. *Neurol* 2006;253:1561-1571.
3. Parisi D, Poullos I, Karkavelas G, Drevelengas A, Artemis N, Karacostas D. Peduncular hallucinosis secondary to brainstem compression by cerebellar metastases. *Eur Neurol* 2003;50:107-109.
4. Nicolai A, Lazzarino LG. Peduncular hallucinosis as the first manifestation of multiple sclerosis. *Eur Neurol* 1995;35:241-242.
5. Geller TJ, Bellur SN. Peduncular hallucinosis: magnetic resonance imaging confirmation of mesencephalic infarction during life. *Ann Neurol* 1987;21:602-604.
6. Maiuri F, Iaconetta G, Sardo L, Buonamassa S. Peduncular hallucinations associated with large posterior fossa meningiomas. *Clin Neurol Neurosurg* 2002;104:41-43.
7. Manfred M, Andermann F. Complex visual hallucinations. Clinical and neurobiological insights. *Brain* 1998;121:1819-1840.
8. Middleton FA, Strick PL. The temporal lobe is a target of output from the basal ganglia. *Proc Natl Acad* 8683-8687.