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A Patient with Amnestic Syndrome and Peduncular Hallucinosis-like Episode Associated with Left Posterior Cerebral Arterial Infarction

Seng-Yun Chung · Seol-Heui Han* Jeong-Eun Sohn*

Department of Pediatrics, Our Lady of Mercy Hospital, Catholic University College of Medicine, Inchon; Department of Neurology, Chungbuk National University Hospital*, Cheongju, Korea

Address for correspondence

Seol-Heui Han, M.D.
Department of Neurology, Chungbuk National University Hospital, 62 Gaeshin-dong, Cheongju 361-711, Korea
Tel: +82-43-269-6372
Fax: +82-43-276-8929
E-mail : shhan@med.chungbuk.ac.kr

'Peduncular hallucinosis' denotes a psychosensorial disorder consisting of multiple visual colored images of brief duration, which usually occur in the evening or in the dark. Typically, the patient is aware that the images are not real. Most cases of peduncular hallucinosis have been described in association with lesions of vascular nature, including thalamic or brain stem ischemia, vasospasm of the perforating brain stem arteries, and pontine or mesencephalic hemorrhage. We describe a patient who presents vivid hallucinatory episode and subsequently develops various neuropsychologic disturbances including amnestic syndrome. The responsible lesion was a large infarct in the left posterior cerebral arterial territory without any evidence of brainstem involvement.

Key Words: *Peduncular hallucinosis, Amnesia, Posterior cerebral arterial infarction*

Peduncular hallucinosis refers to vivid, complex visual perceptions associated with brainstem lesions. Although this rare phenomenon is usually associated with mesencephalic lesion, it also has been reported in ischemic damage and compression of various structures spanning from thalamus to the Vth cranial nerve entry zone. We describe a patient who presents with a similar hallucinatory episode and subsequently develops various neuropsychologic disturbances including amnestic syndrome. The responsible lesion was a large infarct in the left posterior cerebral arterial territory.

CASE REPORT

A 77-year-old man suddenly developed mild weakness of the right leg, dizziness, gait disturbance and dysarthria, causing him to fall down and sustain a rib fracture 2 days before admission. He showed acutely confused mentality and abnormal behavior afterwards. His wife described his nocturnal agitation and misidentification delusion. He imagined an angel in a black robe waiting for him in his bedroom. He insisted that the angel was trying to take him to the next world (peduncular hallucinosis-like episode). Hallucinations occurred when he was relaxed but awake with his eyes shut and waiting to go to sleep. The patient had insulin-dependent diabetes and was managed with 25 units of regular insulin. On admission, his blood pressure was 110/70 mmHg. He was right handed. He was alert but disoriented. Extraocular movement was normal without nystagmus. No cranial nerve palsy was found. Mild right hemiparesis was observed.

He showed a right homonymous hemianopia on visual confrontation test. He could write his name and a short phrase to dictation, but could not read what he had just written. He could not draw a clock, nor copy an interlocking pentagon. For the first two days, he could not recognize the face of her daughter by sight, only to recognize her by voice. On picture naming task, he showed visual perseveration (visualization of afterimage): he tended to name the same objects several times even after he was looking at different objects. He could not accurately reach for visually perceived object. He moved

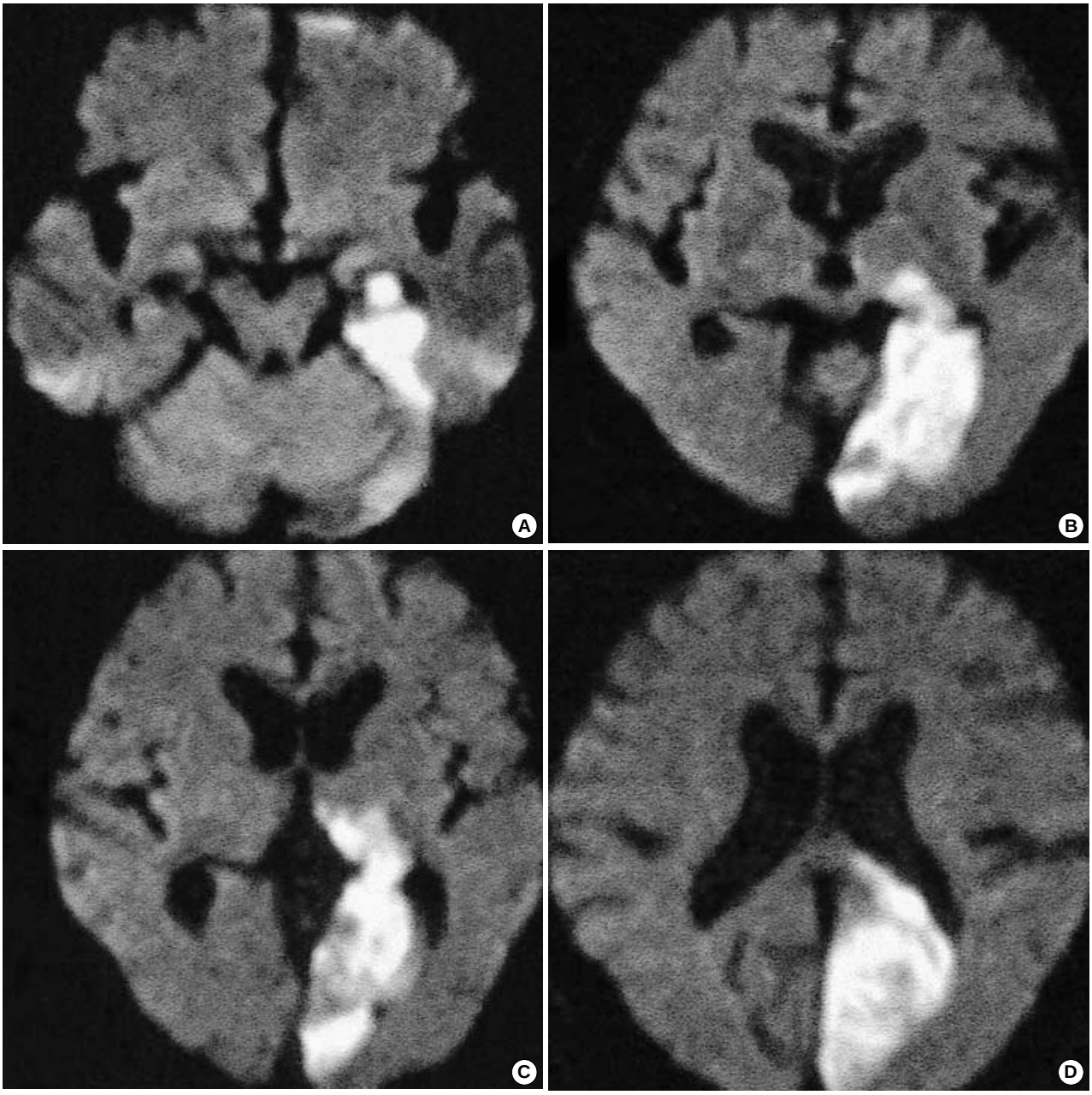

Fig. 1. Diffusion-weighted axial MRI showing acute infarction in the posterior cerebral arterial territory. Increased signal intensities are seen in amygdala and hippocampus (A), geniculate bodies and temporal cortex (B), pulvinar, dorsomedial thalmus and occipital cortex (C), splenium and occipital cortex (D).

a hand into the approximate vicinity of the object and then started searching movements with the hand widely opened. Detailed neuropsychological evaluation revealed alexia without agraphia, color dysnomia, optic ataxia, and profound recent memory defects. Confabulation was also noted. Magnetic resonance imaging demonstrated acute ischemic lesions in the left hippocampus, parahippocampal area, pulvinar, lingual gyrus and splenium of corpus callosum (Fig. 1, 2). No lesion was identified in the mesencephalon. 99mTc-SPECT revealed a large perfusion defect in the left temporo-occipital areas (Fig. 3). Four days after admission, vivid visual hallucination has disappeared. All other neurobehavioral symptoms attenuated afterwards. On follow-up examination three months later, he still showed mild dyslexia and visuo-optic ataxia.

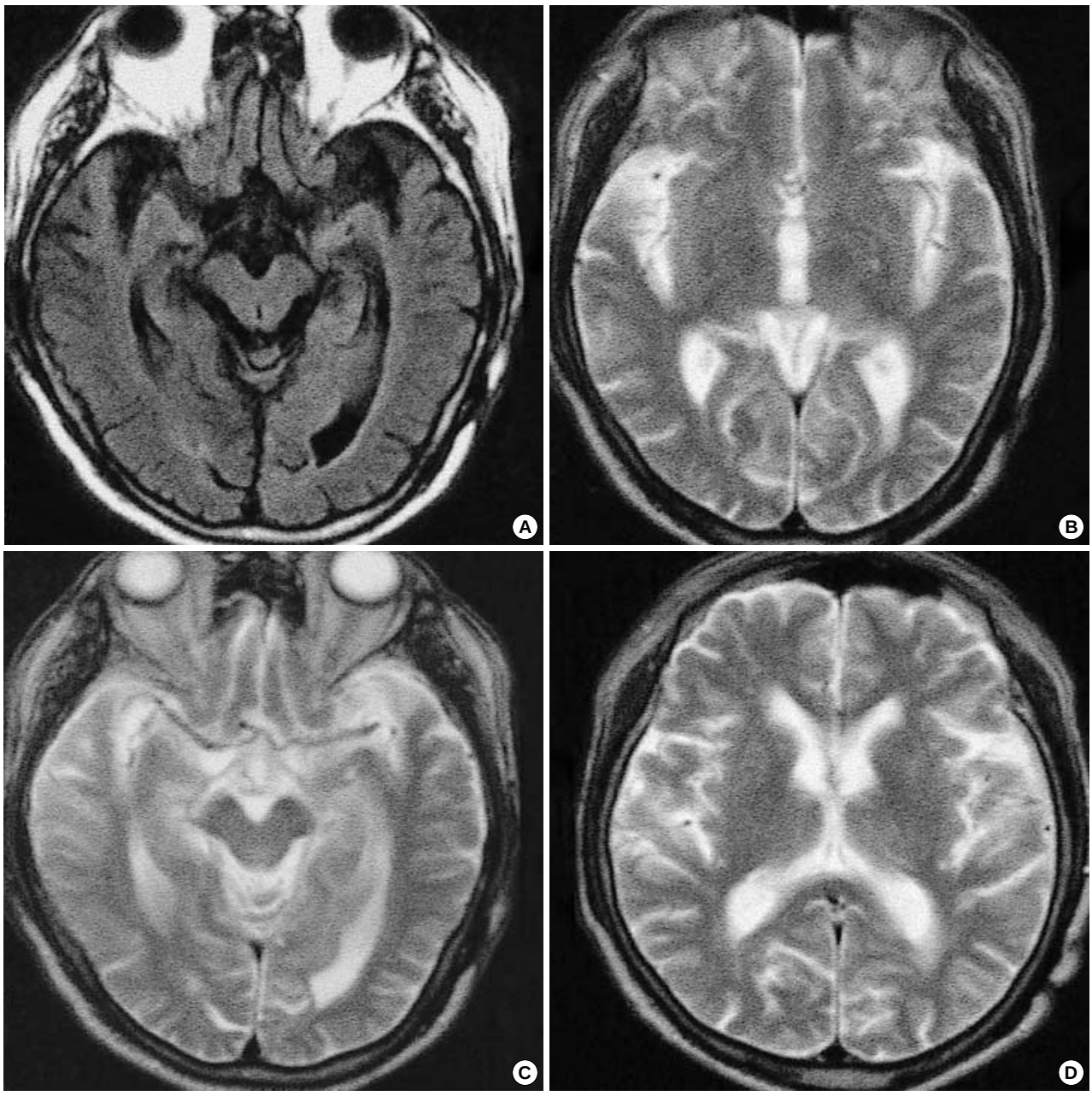

Fig. 2. FLAIR (A) and T2-weighted images (B-D) revealed no chronic responsible lesions seen in Fig. 1.

COMMENTS

Typical clinical presentations in patients with left PCA infarctions include memory or language-related defects. The abnormalities of higher cortical function are invariably related to extrastriate infarction involving the parieto-occipital and/or temporal branches of the left PCA[1].

In our patient, vivid lifelike hallucination suggesting peduncular hallucinosis (PH)-like episode was initial presentation. Besides PH-like episode, the patient showed plethora of neurobehavioral disturbances i.e., alexia without agraphia, color dysnomia, optic ataxia and profound recent memory defects.

The term 'hallucinosis' was coined to refer to alcohol-induced auditory hallucinations, vivid and threatening in nature, accompanied by insight and occurring in states of minimal disorder of consciousness. Visual hallucinations of people or animals accompanied by confusion or disorientation (a dream-like state) have been called "hallucinose pedunculaire" (pedun-

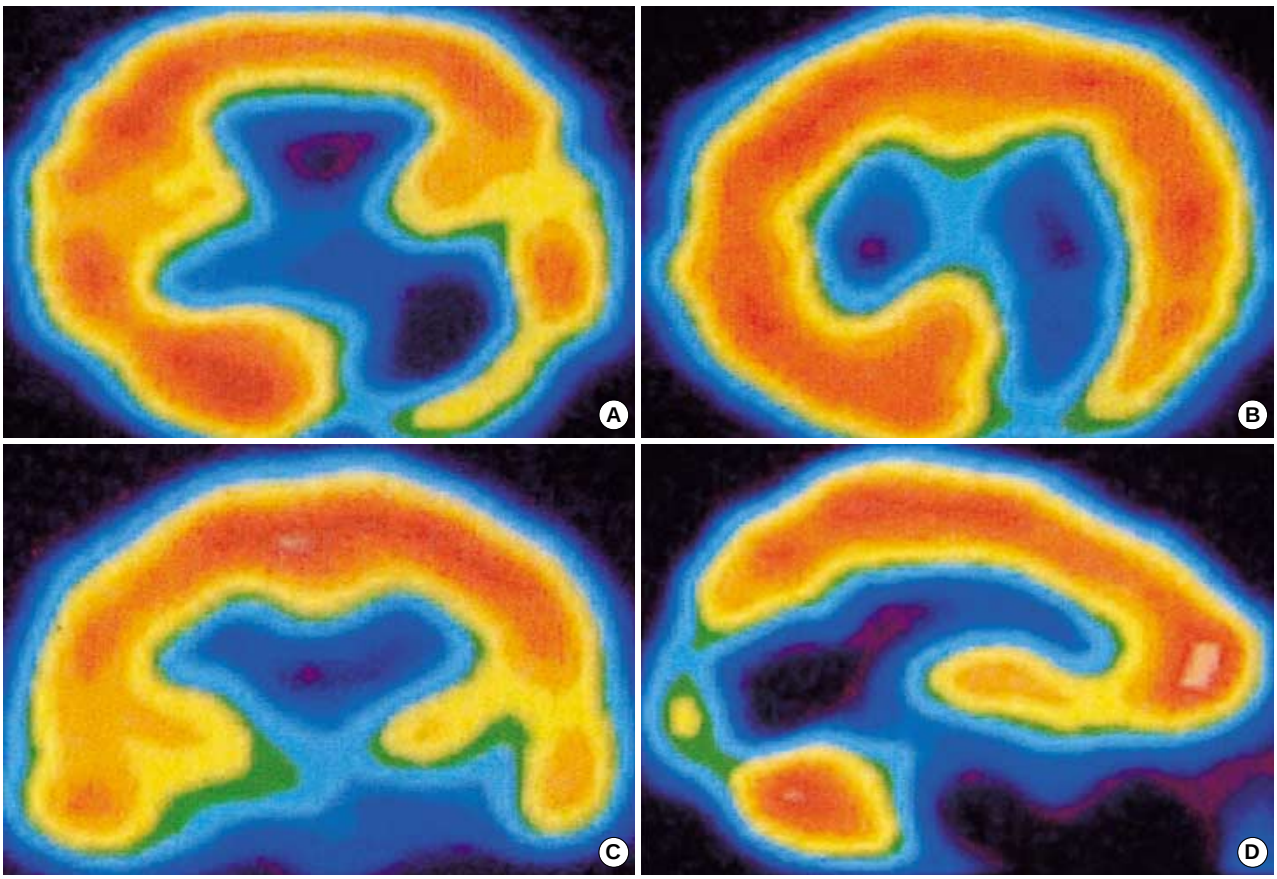

Fig. 3. 99mTc-HMPAO SPECT showing markedly reduced perfusion in left temporo-occipital areas. Axial (A, B), coronal (C), and sagittal (D) images.

cular hallucinosis, PH)[2]. The experiences are usually primarily visual, but may have a auditory and tactile component. They most frequently occur at sundown and are often frightening. As in our patient, these kinds of hallucinations are vivid and lifelike and recognized by the patient as being false perceptions. Some investigators thus have used the term hallucinosis to distinguish them from the usual type of hallucinations. Although the responsible lesion for PH is most commonly localized to the rostral mesencephalon, it also has been reported with lesions of substantia nigra, subthalamic regions, basal ganglia, thalamus, and limbic structures without mesencephalic involvement[3]. In our case, medial thalamus, dorsolateral geniculate nucleus or amygdaloid complex could be the responsible lesion. PH is postulated to be a "release phenomenon" related to damage to the ascending reticular activating system in the midbrain, the thalamus or both[4]. Nevertheless, the transient nature of visual hallucinatory episode seen in our patient is not consistent with the traditional concept of so-called 'pedunculoar hallucinosis'. Both occipital cortex and visual thalamus affected in this patient could be the responsible lesion for the visual hallucination. In their extensive review of complex visual hallucinations, Maford and Andermann[3] pointed out that the hallucinations caused by ischemic lesions in visual cortex are usually transient, lasting days or weeks as in our case. In most cases there is no disorder of arousal or dreams[3]. Charles Bonnet syndrome should be considered in the differential diagnosis of complex visual hallucination. This syndrome is characterized by complex visual hallucinations occurring in the context of ocular pathology causing visual deterioration, most commonly macular degeneration[3]. In the largest study of the condition to date, hallucinations occurred in 10% of patients with severe visual loss, generally in the elderly[4]. If the visual symptom is present longer duration, it is more like peduncualr hallucinosis especially when the patient is drowsy[5]. Palinopsia-like visual illusion, another abnormal positive visual symptom, was observed in this patient. Palinopsia refers to perseveration or the recurrent appearance of a visual image after the stimulus has disappeared. It is usually a transient process, but can persist. The

images are brief and recur periodically in the impaired visual field. This is thought to be a release phenomenon involving lesions of the parietal and occipital lobes most often in the nondoninant hemisphere[6]. It is very unusual to experience a palinopsia-like phenomenon in the left PCA territorial lesion.

It is now known that thalamic lesions can cause severe memory defects. Patients lose the ability to make new memories and may have retrograde amnesia for events before their stroke. Lesions producing memory loss are usually in the distribution of the polar artery and involve the mamillothalamic tract and the ventral portion of the lamina medullaris interna[7]. Lesions involving the medial thalamus and the distribution of the paramedian thalamic arteries can also affect memory as well as visual perception, constructional praxis and temporal orientation[7, 8] Both the left pulvinar, dorsomedial thalamus and hippocampal lesions are related to amnestic syndrome in our patient.

The lesions for optic ataxia could probably be located in the parieto-occipital border zones[9, 10] and according to Perenin and Vighetto[11], tactile apraxia and optic ataxia are explained by deficits of sensory guided motor behavior due to lesions of the posterior cerebral artery territory rather than by unimodal deafferentation or disconnection syndrome.

In summary, our patient showed a unique clinical constellation of full spectrum of posterior cerebral arterial infarct combined with peduncular hallucinosis without mesencephalic involvement.

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