Sequential Appearance and Disappearance of Hemianopia, Palinopsia and Metamorphopsia: A Case Report and Literature Review

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Abstract- We report a case with a sequential appearance and disappearance of homonymous hemianopsia, palinopsia and metamorphopsia in the defective visual field within two days. She had a stroke nine months ago and recovered completely. During that episode, dizziness and unsteadiness happened suddenly and was followed by above-mentioned visual symptoms. Brain image and blood perfusion studies showed an old right occipital lesion with hemodynamic changes. Electroencephalograms performed immediately just after the disappearance of all the above-mentioned symptoms subsided and four months later showed similar findings, some isolated independent sharp waves over bilateral temporal areas, with right posterior extension. These were independent of her visual symptoms. The pathophysiologies of our case could be a sequential hemodynamic change in the right occipital area, from hypoperfusion to transient hyperperfusion and finally to normal perfusion. Potential mechanisms are discussed with literature review.

Key Words: Hemianopia, Palinopsia, Metamorphopsia

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INTRODUCTION

Metamorphopsia is a visual perceptual modification of form, contour or size. Palinopsia is the perseveration of a previously perceived image. Both are considered visual illusion because they occur after exposure to real external visual stimuli[1]. Palinopsia may sometimes happen after a longer latency, such as days or weeks, and could be misinterpreted as a visual hallucination because the patient already forgot previous visual events. These two visual illusions are mainly paroxysmal, appearing as manifestations of epileptic phenomenon, hyperexcitability, psychogenic fantasies, or associated with the progressive evolution or resolution of visual field defect[2-11]. The underlying mechanism remains uncertain. In this report, we describe a patient with a sequential appearance and disappearance of homonymous hemianopsia, palinopsia and, then, metamorphopsia in the defective visual field. To our knowledge, no such has been reported. The possible pathophysiology is discussed with literature review.

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CASE REPORT

The patient was a 59-year-old right handed, illiterate woman. She had a stroke nine months ago, which presented as dizziness and unsteady gait without focal weakness. The symptoms subsided completely within one week, though the actual lesion site was not defined. She had hypertension for several years without regular medication. On one morning, while she was resting on a chair after coming back from the market, she suddenly felt dizzy. When she tried to walk into the room, she felt unsteady though no focal weakness or numbness was noted at that moment. Her speech became slurred and her response was dull. She was brought to our ER immediately. On arrival, she was fully oriented but slow in response. Neurological examination was normal except for left hemiparesis (R/L: 5/4) without obvious discrepancy between upper and lower limbs. Her visual field was not checked at ER. She was admitted under the impression of right supratentorial infarction.

On admission, her vital signs were stable. She was orientated but slow in response. Neurological examination showed normal reactive pupils, adequate visual acuity and a full range of eye movements. The confrontation test showed dense left homonymous hemianopia. She had slurred speech. Yet, her language functions, including comprehension, repetition, naming and reading, were grossly normal. Writing could not be tested since she was illiterate. Calculation was partially impaired. She could recognize color, face and objects correctly. No left-sided neglect could be identified by the line bisection performance, selection of three test or figure copying. In the double simultaneous stimulation test with auditory and tactile stimuli, inattention to the left side was noted. Inattention to the visual stimulation could not be confirmed in the presence of the left homonymous hemianopia. The results of other routine neurological examinations were normal except a wide-based gait and an unsteady tandem gait. Serum biochemistry tests showed that renal and liver functions, sugar and electrolytes were normal. No leukocytosis, anemia or pyuria was noted.

On the next day, the visual field defect recovered. However, visual perseveration appeared. The vivid image of the examiner's finger persisted in her left visual field for about 3 minutes after the fingers were gone. She reported that the fingers were waving, not on a stationary position. On the third day, she described that the phenomenon of visual perseveration disappeared and, instead, she saw the left edge of bed tilting downward and the left railing of the bed became far from her. She felt that she was going to fall down from the left side. In addition , the quilt became triangular and she tried in vain to fold it back as a square. Her daughter and nurses around her did not see the deformed objects as she described. This phenomenon persisted for about twelve hours. Thereafter, she never had similar experiences. The double simultaneous stimulation test performed every day during her hospital stay revealed a gradual improvement day by day. Audiometric examination reported normal hearing.

Brain MRI, performed on the next day after the

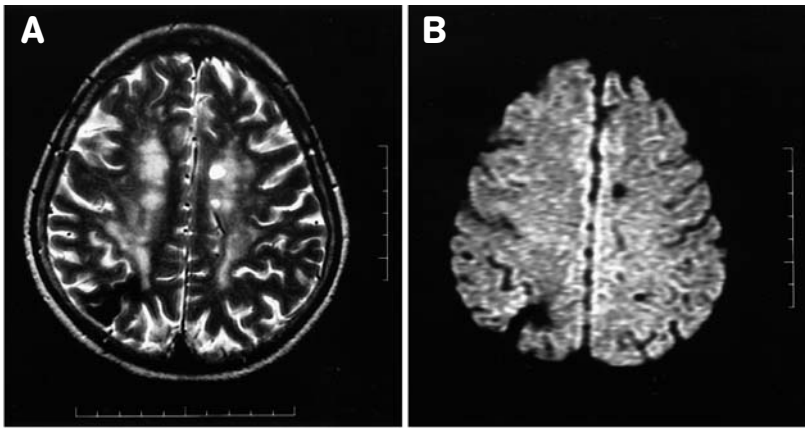

Figure 1. (A) T2-weighted brain MRI image showing a lesion in the right occipital area and (B) diffusion scan image showing no acute lesion in the corresponding area.

onset of symptoms, showed no recent infarct but an old infarct in right occipital region (Fig. 1). EEG performed twice, on the third and sixth day after the onset of symptoms, showed a few isolated independent sharp waves over bilateral temporal areas, with right posterior extension. Tc99 HMPAO single photon emission computed tomography (SPECT) performed on the eighth day revealed hypoperfusion on the right occipital and left temporal areas (Fig. 2A). Visual evoked potential and brainstem auditory evoked potential studies performed on the same day were normal. On discharge, she was free of neurological deficits. SPECT and EEG studies were repeated four months later. The EEG abnormalities remained. The hypoperfusion on the right occipital and left temporal areas on the SPECT was more prominent (Fig. 2B). The patient was followed up for four months and reported that the abnormal visual experience never happened again.

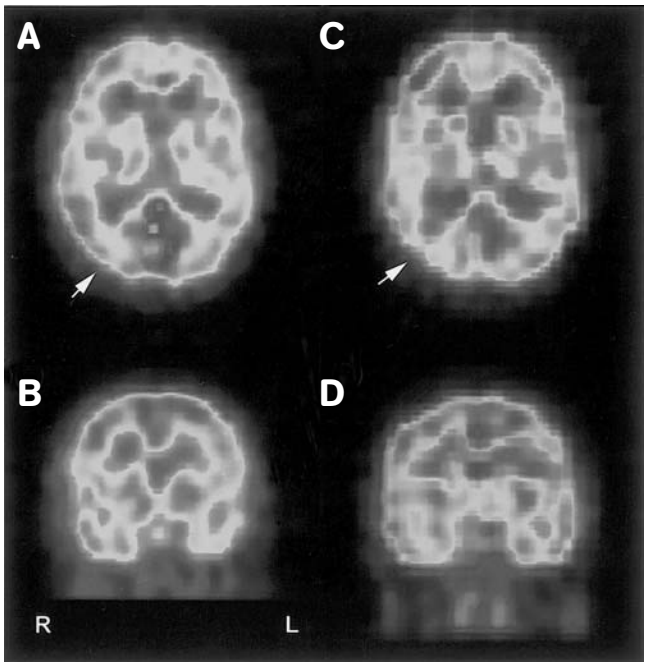

Figure 2. Images of SPECT taken just after the symptoms subsided (A and B) and four months later (C and D). The hypoperfusion of right occipital area (pointed by an arrow) in the acute stage was more prominent in 4 months later.

DISCUSSION

Etiology

Many etiologies were implicated for causing palinopsia (Table 1). Palinopsia may also appear in normal persons as the so-called physiological after-image. Cases with optic neuritis have been reported, whose visual illusion was confined to one eye[6,10]. The etiologies of intracranial focal lesions include space-occupying lesions, such as neoplasm[11-13], cyst[2] abscess[14-15], trauma[16], arterio-venous malformation[17], demyelination

Table 1. *Etiologies of palinopsia*

Non-pathological condition[10]	**Diffuse or poor localized brain lesions**
Diseases confined to the eye or optic nerve	Drug-related
Hypertensive retinopathy[35]	Marijuana
Optic neuritis[6,10]	Mescaline[52]
After laser treatment of DM macular edema[10]	Lysergic acid diethylamine[27]
Leber's hereditary optic neuropathy[10]	Interleukin-2[26]
Focal brain lesions	Trazodone[25]
Stroke (infarct or hemorrhage)[5,7]	Clomiphene citrate
Neoplasm[11-12,35]	Nefazodone
Cyst[2]	CO intoxication
Abscess[14-15]	Creutzfeldt-Jacob disease[24]
Trauma[16]	Nonketotic hyperosmotic syndrome[23]
AVM[17, 35]	Psychiatric disease
Parasite[19]	Schizophrenia[8]
Demyelination diseases, including MS[6,10,18]	Psychotic depression[4]
Seizure	**Charles Bonnet syndrome**[53]
Occipital[9]	
Temporal[2,7]	
PLED[11]	
Migraine[22]	

disease[6,10,18], parasite infection[19], strokes (infarct[20,21] and hemorrhage[5]), and functional deficits, such as migraine[22] and seizure[2,7,9,11]. In the category of diffused neurological diseases, which usually present the visual illusion in a global manner without lateralization, non-ketotic hyperosmolarity syndrome[23], schizophrenia[8], psychotic depression[4] and Creutzfeldt-Jacob disease[24] have been reported. It can also be the adverse effect of some drugs, such as trazodone[25], interleukin 2[26], and lysergic acid diethylamide (LSD)[27].

Localization

The locations responsible for visual illusion also varied. It has been reported in healthy persons[10] and in patients with lesions ranging from the eyes to the cerebral cortex. In the category of intracranial focal lesions, the visual illusion is often confined to the contralateral defective visual field. The visual field defect indicates the dysfunction of visual pathway. Pomeranz et al.[10] reported a case of optic neuritis presenting visual illusion in the process of recovery from blindness. The illusive image was in the entire visual field of the affected eye. Sensory deprivation, however is not absolutely necessary for the generation of those symptoms. Young et al.[11] reported a case of right parietal astrocytoma with episodic palinopsia and metamorphopsia in left visual field but there is no visual field defect throughout the course. When the deprivation of vision is not confined to one half-field, illusive image may not be lateralized, either. Cases with diffused or poor localized neurological disease usually have neither lateralized visual illusion nor visual field defect. Their illusive image could appear in the entire visual field.

The most common location of intracranial focal lesions is the occipital lobe of non-dominant hemisphere, including primary and associated visual cortices such as areas 17, 18 and 19. The lesions may extend to areas 21, 22, 37 and 39, optic radiations and mesial fusiform gyrus[28]. Cases with lesions in the dominant hemisphere have also been reported[12,19,21]. Our case, who had an old infarct in the occipital area of non-dominant hemisphere with the illusive images appearing in the partially defective visual field, was similar in location to those reported previously[2,5,11,20,21]. Vaphiades et al.[28] observed that patients with lesions in the MCA territory did not have metamorphopsia or palinopsia. This finding implied that more anteriorly located visual association areas containing stored representation of visual features were necessary for the production of illusive images. On the other hand, an intact visual pathway leading to the visual cortex might be necessary for the production of visual illusion.

Most of the cases with metamorphopsia have their lesions in the eyes or optic nerves[29-34]. Only a few cases with brain lesions have been reported[11,39-49].

Mechanisms

Many pathophysiological mechanisms have been postulated to explain the visual illusion. Four major possible mechanisms of palinopsia are physiological after-image, hyperactivity in vision related areas, epilepsy and drug-induced phenomenon (Table 2). Though some researchers[35] also include psychogenic visual hallucination as one of the mechanisms, visual hallucination and illusion are contradictory by definition and will not be discussed further.

The physiological after-image is essentially a positive contrast of the primary stimulus and is more visible after light stimulation. It is characterized by (1) the illusive image is perceived whether the eyes are open or not, (2) there is no latency between the original and the illusive images, (3) the duration of the illusive image is

Table 2. *Proposed pathophysiologies of palinopsia*

Pathophysiology	Physiological after-image	Hyperactivity	Epileptic phenomenon	Drug effect
Image size	Whole field	Focal / Whole field	Hemi-field	Whole field
Reversed color	Yes	No	No	No
Latency	No	Long / Short	Long / Short	Short / No
Duration	Short	Variable	Variable	Variable
Attack continuity	Continuous	Episodic	Episodic	Continuous
Visual field defect	No	Yes / No	Yes / No	No

short, and (4) the color is complementary to the original image. Blythe et al.[18] have reported a case of abnormally prolonged after-image, which appeared in the whole visual field with absence of visual field defect, dyschromatopsia or localized cortical lesion. Although there was a focal EEG abnormality, it was independent of the visual phenomenon. The mechanism of the abnormally prolonged physiological after-image could be a general lack of the mechanism responsible for signaling stimulus offset, or a central adaptation phenomenon.

Seizure as a mechanism received much more attention. It was postulated that the abnormal neuronal discharge caused the prolonged existence of images[2,7,9,11]. Müller et al.[9] reported three cases of palinopsia. These three patients had metastatic neoplastic lesion or infarction on the right occipitotemporal region and left hemianopia. Their EEG showed epileptiform discharges from the lesion site with or without secondary generalization. The symptoms of these three cases disappeared after being treated with phenytoin or carbamazepine. Lefebre and Kölmel[7] reported a case with a right parieto-occipital astrocytoma having delayed palinopsia in her defective upper left quadrant visual field six months after excision of the tumor. EEG recorded during the onset of symptoms showed epileptiform discharge in right temporo-parietal area. The close relationship between the epileptiform discharge and the symptoms indicated that the visual illusion was due to seizure. Another case reported by Young et al.[11] also had a right parietal astrocytoma. The palinopsia and metamorphopsia occurred one month before she received surgical intervention and radiation therapy. The EEG revealed periodic lateralized epileptiform discharge (PLED) with a repetitive rate of 1 to 1.5 Hz in the right parietal area, compatible with the localization of the astrocytoma. Her symptoms were only present when she had PLEDs. In contrast to the cases mentioned above, she did not have visual field defect throughout the course. There are also reports[36-37] describing delayed-onset palinopsia and palinacousis as an epileptic phenomenon of the temporal lobe. The long latency indicated an errant recall of long-term or short-term visual memory.

In addition to seizure, hyperactivity is another potential mechanism. Cummings et al.[2] suggested that palinopsia was a unique phenomenon of releasing stored memory of various sensory modalities resulting from loss or suppression of normal visual input. However, he did not regard it as a seizure phenomenon and thought the visual field defect should be an absolute pre-existed factor. Maillot et al.[38] also suggested that palinopsia was due to a dysfunction of the visual memory, rather than the visual sensing system and this dysfunction might be related to the hyperactivity of the visual buffer. Hemodynamic change is a frequent cause of disinhibition in sensory deprivation. The case reported by Hayashi et al.[5] had a right occipital hematoma and presented with transient palinopsia in the contralateral visual field in the recovery process. SPECT showed a hyperperfusion ring surrounding the hematoma in the period of symptoms. The hyperperfusion ring became hypoperfused after the symptoms disappeared. So the authors indicated that the hyperperfused area surrounding the hematoma might be a hint of cortical instability and excited neuronal activity in the area during recovery from hemianopsia.

There are also reports[25-27] discussing the effect of drugs on inducing visual illusion. Trazodone and LSD inhibit serotonin reuptake and interfere the homeostasis of serotonin level in the brain. Yet, how the serotonin reuptake inhibitors are related to the visual illusion has not been addressed.

The pathophysiology of metamorphopsia has been less well studied. For most cases having lesions in the eyes and optic nerves, the mechanism is believed to be the distortion of retina or optic nerve[30-31]. For those cases with abnormalities suspected to reside in the brain[39], epilepsy[11,40-41], stroke[42-45], migraine and hemodynamic change[46-49] are proposed candidates. It is difficult to classify metamorphopsia as a positive or a negative symptom and it is not clear whether metamorphopsia is caused by hyperperfusion or hypoperfusion since both have supporting reports. There are also reports relating metamorphopsia to Epstein-Barr virus infection. The exact location and mechanism are unknown[50-51].

Our case had a sequential appearance and disappearance of visual field defect, palinopsia and metamorphopsia in the defective visual field. In contrast to Hayashi's case, whose SPECT showed a ring hyperperfusion dur-

ing the symptomatic stage and hypoperfusion in the symptom-free period, our case presented hypoperfusion in SPECT just after the symptoms subsided and returned to normal perfusion four months later. It is possible that we only captured the hypoperfusion stage after the ring hyperperfusion stage, and the symptoms recovered while there was still relative hypoperfusion. The evolving clinical course from negative symptoms (visual field defect), to positive symptoms (palinopsia), to metamorphopsia and finally to the complete remission of symptoms within 2 days strongly suggests a sequential hemodynamic change in the right occipital area, from hypoperfusion to transient hyperperfusion and finally to normal perfusion.

Though the EEG showed independent sharp waves in bilateral temporal areas, these EEG abnormalities were stationary. Therefore, epilepsy was less likely to be the sole underlying mechanism. A combined effect of epileptic hyperexcitability and hemodynamic change cannot be completely ruled out.

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