

# Acute exacerbation of multiple sclerosis presenting with facial metamorphopsia and palinopsia

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### Abstract

We discuss the case of a patient with a known history of relapsing-remitting multiple sclerosis (MS) who presented with the isolated complaint of altered visual perception in the absence of abnormalities on ophthalmological examination. To the best of the authors' knowledge, this is the first documented case of both facial metamorphopsia and palinopsia occurring as the symptoms of demyelinating brain lesions consistent with an acute MS exacerbation. These symptoms appear to be related to active demyelination that either involved the optic radiations in the visual pathway or the visual association area in the temporo-occipital region of the left hemisphere.

#### **Keywords**

demyelination, metamorphopsia, palinopsia

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## **Case presentation**

Our patient is a right-handed woman with a 2-year history of relapsing-remitting multiple sclerosis (MS) on treatment with interferon beta-1a injections, who presented at the age of 32 with altered visual perception. She reported that she experienced distorted faces in her right field of vision. When looking at any face, the left side of the face that she perceived in her right visual field appeared distorted or elongated. In particular, she said that peoples' eyes appeared stretched out in an almost ovoid shape on this side of their face. See Figure 1. She did not report such distortions for anything other than faces.

She also described seeing persistent translucent 'afterimages' of previously witnessed visual images. For instance, she would perceive the image of a person seated on a chair for several seconds to minutes after the person had stood up and walked away from the chair. Our patient had no prior history of other neurological disorders, optic neuritis, seizures, trauma, or HIV risk factors. There was no family history of neurological diseases or autoimmune disorders.

Ophthalmological examination showed symmetric and appropriately reactive pupils, normal visual acuity, full perception in all visual fields, full range of extraocular movements, and normal fundoscopic examination. Neurological examination was unremarkable aside from difficulty with performing tandem gait. MRI performed during the presence of these symptoms showed an extensive area of T2-hyperintense and T1-hypointense signal with enhancement in the left periatrial region extending into the occipital subcortical white matter with minimal mass effect as well as a second enhancing lesion in the left frontal area. See Figure 2. Additional non-enhancing smaller lesions were noted in the bilateral periventricular, pericallosal, and corpus callosal regions. CSF studies were remarkable for positive oligoclonal bands, elevated IgG index of 0.82 (normal 0.28–0.66) and otherwise normal studies including cell count, glucose, protein, cultures, and cytology.

Our patient's visual symptoms persisted for several weeks, after which they began to improve spontaneously without steroid treatment but never to her reported baseline, although she continued to experience these phenomena at various intervals without identifiable triggers.

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**Figure 1.** Patient's illustration of her visual perception of faces. The left side of the face that she perceived in her right visual field appeared distorted and horizontally elongated. The opposite side appeared normal.



**Figure 2.** MRITI-weighted axial image with gadolinium shows irregular enhancement in the left periatrial region extending into the occipital subcortical white matter, indicating active demyelination.

## Discussion

Metamorphopsia is the visual perceptual modification of form, contour, or size. Facial metamorphopsia is a type of metamorphopsia specific for faces while palinopsia is the perseveration of a previously perceived image after removal of the visual stimulus.<sup>1</sup> These positive visual symptoms may occur in the absence of visual-field deficits.<sup>2</sup> These entities are categorized as visual illusions because they occur after exposure to external visual stimuli, thereby differentiating them from visual hallucinations. They have been reported with ocular disease, focal brain lesions including ischemic insults, toxic-metabolic processes, as well as psychiatric disease. The proposed mechanisms of palinopsia include physiological afterimage, hyperactivity in vision related areas, epilepsy, and drug-induced phenomenon, while the proposed mechanisms of metamorphopsia include distortion of the retina or optic nerve, epilepsy, migraine, and hemodynamic changes.3

To the best of the authors' knowledge, our case is the first documented report of a patient with both facial metamorphopsia and palinopsia occurring as the initial presenting symptoms of demyelinating brain lesions consistent with an acute exacerbation of MS. Although there has been one published case of palinopsia associated with the recovery from optic neuritis in a patient who later developed MS, this patient had no abnormalities on MRI brain imaging at the time she developed palinopsia.<sup>4</sup> When our patient initially presented with her visual symptoms, MRI brain demonstrated an actively enhancing lesion in the left temporo-occipital region. In the absence of alternative etiologies, we posit that our patient's positive visual symptoms were related to active demyelination that either involved the optic radiations in the visual pathway or the visual association area in the temporo-occipital region of the left hemisphere. In addition, our patient's complaints were associated with active demvelination in the left hemisphere, which leads us to consider that these symptoms are not localized to dysfunction of the nondominant hemisphere.

The current literature has attempted to elucidate the etiologies and cortical localizations associated with metamorphopsia and palinopsia.<sup>1,4,5,6</sup> These visual phenomena typically arise from altered function in the temporo-occipital region of the non-dominant hemisphere with proposed specific involvement of the primary and associated visual cortices, optic radiations, areas 21, 22, 37, and 39, and mesial fusiform gyrus.<sup>1,7</sup> However, rare cases have been described when these illusions localize to the dominant hemisphere. Shiga et al. presented a case of left putaminal hemorrhage followed by metamorphopsia in the right visual hemifield, presumably due to involvement of the left optic radiations.8 Imai et al. reported a case of abrupt onset of right-sided metamorphopsia occurring in the context of an acute infarct occurring between the retrosplenium and the left cingulate gyrus.9 Palinopsia due to dominant hemispheric dysfunction has been reported by Ritsema et al., who described two cases of palinopsia caused by left hemispheric structural lesions of the posterior visual pathway.<sup>2</sup>

Our case illustrates the importance of evaluating visual complaints even in the absence of visual field or visual acuity deficits on examination and adds to the spectrum of neurological symptoms that may occur in patients with MS.

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#### **Conflict of interest**

The authors declare no conflicts of interest in preparing this article.

#### References

 Sun Y and Lin CK. Sequential appearance and disappearance of hemianopia, palinopsia and metamorphopsia: a case report and literature review. *Acta Neurologica Taiwanica* 2004; 13: 77–83.

- Ritsema ME and Murphy MA. Palinopsia from posterior visual pathway lesions without visual field defects. *J Neuro-Ophthalmol* 2007; 27: 115–117.
- Bender MB, Feldman M and Sobin AJ. Palinopsia. Brain 1968; 91: 321–338.
- Pomeranz HD and Lessell S. Palinopsia and polyopia in the absence of drugs or cerebral disease. *Neurology* 2000; 54: 855–859.
- Heo K, Cho YJ, Lee SK, Park SA, Kim KS and Byung IL. Single-photon emission computed tomography in a patient with ictal metamorphopsia. *Seizure* 2004; 13: 250–253.
- Meadows JC and Muro SF. Palinopsia. J Neurol Neurosurg Psychiatry 1977; 40: 5-8.
- Vaphiades MS, Celesia GG and Brigell MG. Positive spontaneous visual phenomena limited to the hemianopic field in lesions of central visual pathways. *Neurology* 1996; 47: 408–417.
- Shiga K, Makino M, Ueda Y and Nakajima K. Metamorphopsia and visual hallucinations restricted to the right visual hemifield after a left putaminal haemorrhage. *J Neurol Neuro*surg Psychiatry 1996; 61: 420–421.
- Imai N, Nohira O, Miyata K, et al. A case of metamorphopsia caused by a very localized spotty infarct. *Clin Neurol* 1995; 35: 302–305.