Case Report

Bilateral Optical Nerve Atrophy Secondary to Lateral Occipital Lobe Infarction

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Abstract

Purpose: To report a phenomenon of optical nerve atrophy secondary to lateral occipital lobe infarction.

Methods: Two successive patients with unilateral occipital lobe infarction who experienced bilateral optical nerve atrophy during the follow-up underwent cranial imaging, fundus photography, and campimetry.

Results: Each patient was diagnosed with occipital lobe infarction by cranial MRI. During the follow-up, a bilateral optic atrophy was revealed, and campimetry showed a right homonymous hemianopia of both eyes with concomitant macular division.

Conclusion: Bilateral optic atrophy was related to occipital lobe infarction, and a possible explanation for the atrophy was transneuronal degeneration caused by occipital lobe infarction. (***Eye Science 2013; 28:92-94***)

Keywords: optic atrophy; occipital lobe infarction; transneuronal degeneration

Introduction

The occipital lobe is the higher visual center located at the back of the hemisphere. Occipital lobe infarction occurs when the blood supply from the cortical branch of the posterior cerebral artery is interrupted. It is clinically characterized by ocular manifestations, including bilateral impaired vision, visual hallucination, and bilateral homonymous hemianopia with or without sparing of macula, with generally normal pupils and optic disks. In clinical practice, we encountered two successive patients with unilateral occipital lobe infarction who experienced bilateral optical nerve atrophy during the follow-up.

Case reports

Case 1: the patient was a 46-year-old man who experienced a sudden onset of visual decrease in the bilateral eyes, accompanied by vertigo, on December 18th 2008. There was no eye redness, eye pain, diplopia, or abnormal limb movement. A cranial MRI revealed massive softening lesions of the left occipital lobe (Figure 1), and the patient was diagnosed with left occipital lobe infarction. He had no previous history of hypertension, diabetes mellitus, or ocular diseases. On March 13th 2009, his bilateral best corrected visual acuity was 0.5, pupils were 3 mm in diameter, and the direct and indirect light reflexes were normal. The relative afferent pupillary defect (RAPD) was negative. Fundus photography revealed a bilateral optic atrophy (Figure 2). Campimetry showed a right homonymous hemianopia of both eyes, with concomitant macular division (Figure 3).

Case 2: the patient was a 30-year-old man who experienced sudden dizziness, headache, restlessness, and subsequent lethargy and unconsciousness on November 23rd 2007. A head CT scan revealed hematoma in left frontal lobe and temporal lobe (Figure 4); the patient was diagnosed with left intra-cerebral hematoma. He had no previous history of hypertension, diabetes mellitus, or ocular diseases. On December 3rd 2007, the patient underwent left craniotomy for hematoma evacuation and hemicraniectomy. At 20 days after the surgery, the patient regained consciousness and reported bilateral impaired vision, more prominently in the left eye, without bloodshot eye, ophthalmalgia, or diplopia. On May 7th 2008, an MRI scan revealed massive

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malacia in left occipital lobe (Figure 5), and the patient was therefore diagnosed with left occipital lobe infarction secondary to intra-cerebral hematoma. On May 10th 2008, the patient underwent skull neoplasty with an autogenous bone flap. On March 9th 2009, a repeated examination showed that best corrected visual acuity was 0.2 in the right eye and 0.1 in the left eye; the diameter of the pupil was 3 mm in both eyes, and pupils were well responsive to light; RAPD was negative; both optic disks were pale in color, with well-defined boundary (Figure 6); ocular movements were not impaired; and camp-imetry suggested right homonymous hemianopia in both eyes, accompanied by macular splitting (Figure 7).

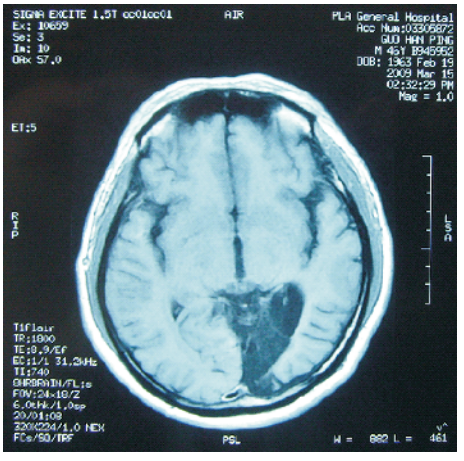

Figure 1 T1 flair image of the cranial MRI revealed massive softening lesions of the left occipital lobe in case 1.

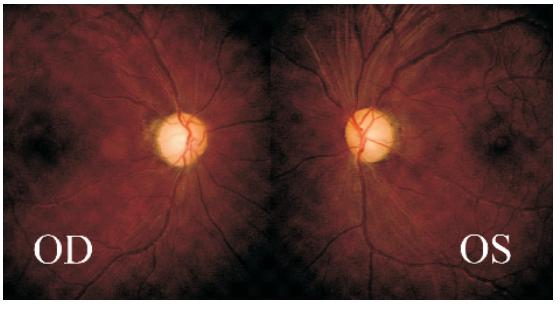

Figure 2 Fundus photography revealed a bilateral optic atrophy in case 1.

Discussion

Based on the patient data described above, these patients had unilateral occipital lobe infarction and bilateral optical nerve atrophy. Ordinarily, occipital lobe infarction will not cause optical nerve atrophy

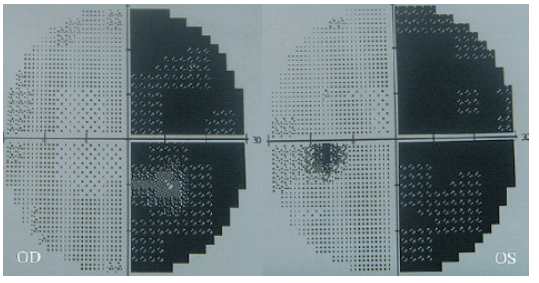

Figure 3 A right homonymous hemianopia of both eyes with concomitant macular division in case 1.

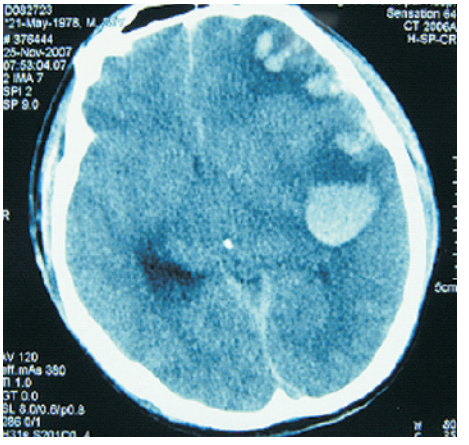

Figure 4 A head CT scan revealed hematoma in left frontal lobe and temporal lobe in case 2.

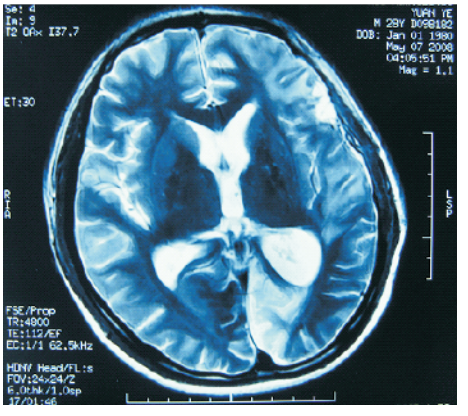

Figure 5 MRI scan revealed massive malacia in left occipital lobe in case 2.

in a direct manner. However, we believed that the optical nerve atrophy in these two patients was related to occipital lobe infarction for the following reasons: ① visual field defect of homonymous hemianopia that was consistent in both eyes, together with negative RAPD, suggesting lesions in posterior optic pathway; ② in both patients, no evidence suggested lesions in the retina, optical nerve, optic chiasma, or anterior optic tract; ③ cranial MRI pro-

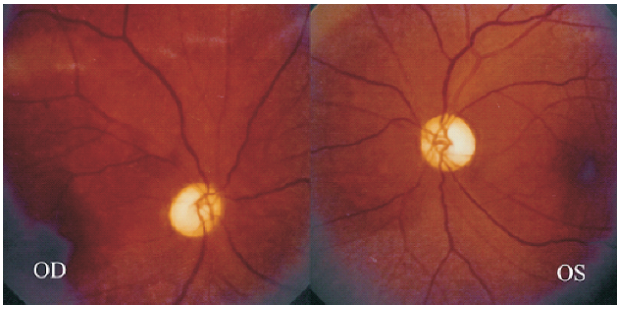

Figure 6 Fundus photography revealed a bilateral optic atrophy in case 2.

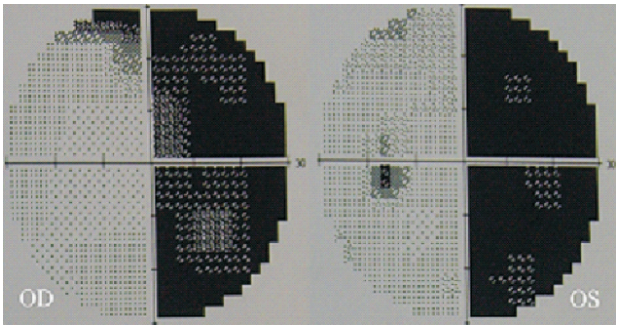

Figure 7 A right homonymous hemianopia in both eyes, accompanied by macular splitting in case 2.

vided direct evidence of unilateral occipital lobe infarction that agreed with visual field defects; ④ both patients might have, to a varied extent, intra-cranial hypertension, particularly in case 2, who had occipital lobe infarction secondary to intra-cerebral hematoma. In this scenario, intra-cranial hypertension was caused by hydrocephalus and intra-cerebral hematoma, and generally lasted for a short period of time. In addition, optical nerve atrophy caused by intra-cranial hypertension was mostly secondary to papilledema, and thus should be accompanied by relevant ocular fundus changes, and the visual field defect was not necessarily consistent homonymous hemianopia. Therefore, the evidence was insufficient to support optical nerve atrophy ascribable to intracranial hypertension in these two patients.

Currently, the mechanism of the optical nerve atrophy caused by occipital lobe infarction is still unclear. Occipital lobe infarction results in direct damage to the neurons in the visual cortex and the optic radiation, possibly even in the lateral geniculate body, but because it has no direct impact on retinal ganglion cells, optical nerve atrophy is not expected. However, numerous animal studies have demonstrated that destruction or resection of the occipital lobe cortex in monkeys[1] and cats[2] could result in neuron degeneration in the lateral geniculate body, and also degeneration and loss of retinal ganglion cells, a process termed "transneuronal degeneration." In general, transneuronal degeneration infers that when impairment of neuron occurs, the neighboring neurons (or even more distant neurons) may also be involved. Transneuronal degeneration has also been seen in glaucoma[3], retinitis pigmentosa[4], and in the substantial nigra-striatum[5], hearing[6] and olfactory[7] conduction pathways. Hence, a possible explanation for the optical nerve atrophy in these two patients was transneuronal degeneration caused by occipital lobe infarction. However, further work is necessary to confirm whether these changes arise from transneuronal degeneration.

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