Binasal hemianopia caused by bilateral optic perineuritis due to sarcoidosis

**ARTICLE INFO**

**Keywords**
Binasal hemianopia
Optic perineuritis
Sarcoidosis

Dear Editor,

Optic perineuritis (OPN) is a rare orbital inflammatory disease that affects the optic nerve sheath. Although many cases of OPN are idiopathic, various infectious and autoimmune diseases have been reported as a cause of secondary OPN, such as syphilis, giant cell arteritis, Behçet’s disease, granulomatosis with polyangiitis, Crohn’s disease, anti-myelin-oligodendrocyte glycoprotein antibody syndrome, and sarcoidosis [1–5]. Visual field abnormalities in OPN include enlarged blind spots as well as arcuate and paracentral scotomas [1,2], whereas binasal hemianopia (BNH) has not been reported. Unlike homonymous and bitemporal hemianopia, BNH is a rarely encountered neuro-ophthalmological finding. Since visual signals from the nasal visual fields do not cross but pass through the lateral nerve fibers at the optic chiasm, bilateral temporal optic nerve fiber lesions or uncrossed optic nerve fibers should be considered as responsible lesions of BNH [6,7]. Here, we describe a case of BNH secondary to bilateral OPN presumably caused by sarcoidosis, which to our knowledge, is the first such report in the literature.

1. Case report

A 59-year-old female with no significant past medical history presented to an ophthalmologist with vision deficit lasting two years and was referred to our hospital. Upon examination, her visual acuity was 0.1 and 0.8 in the right and left eye, respectively. Both pupils were equal; however, the response to light was sluggish with a relative afferent pupillary defect (RAPD) in the right eye. Confrontation tests revealed binasal visual field defects. Goldmann visual fields (GVF) confirmed the presence of incomplete binasal visual field defects (Fig. 1A). The anterior segment and funduscopic exams revealed no signs of uveitis, and no swelling or color change of the discs (Fig. 1B). The central critical flicker frequency (CFF) was 14.2 Hz and 26.3 Hz in the right and left eye, respectively. Ocular coherence tomography (OCT) showed improved binasal visual field defects. Goldmann visual fields (GVF) confirmed the presence of incomplete binasal visual field defects (Fig. 1A). The anterior segment and funduscopic exams revealed no signs of uveitis, and no swelling or color change of the discs (Fig. 1B). The central critical flicker frequency (CFF) was 14.2 Hz and 26.3 Hz in the right and left eye, respectively. Ocular coherence tomography (OCT) showed a reduction in macular retinal thickness in both eyes, but more severely in the right eye. Laboratory tests, which included tests for anti-nuclear antibody, anti-neutrophil cytoplasmic antibody, angiotensin-converting enzyme, syphilis serologic test, and anti-aquaporin 4 antibody were all negative. Bilateral hilar lymphadenopathy was detected on chest radiography. The cerebrospinal fluid analysis revealed protein elevation at 71.5 mg/dL and was positive for oligoclonal bands. Bronchoalveolar lavage revealed an elevation of CD4/CD8 T lymphocyte ratio at 4.49. Gadolinium-enhanced brain magnetic resonance imaging (MRI) showed circumferential enhancement of the bilateral optic nerve sheaths (Fig. 1C), suggesting OPN, while no compression lesion or enhancement was found at optic nerves, optic chiasm, and post-chiasmal visual pathways. In addition, there are no typical lesions consistent with multiple sclerosis on brain MRI or long spinal cord lesions on spinal MRI suggesting neuromyelitis optica spectrum disorder. Gallium scintigraphy revealed accumulation in the mediastinal lymph nodes (Fig. 1D). Biopsy from the mediastinal lymph node showed non-caseating epithelioid granulomas and multinucleated giant cells (Fig. 1E, F). Thus, the probable diagnosis of bilateral OPN secondary to sarcoidosis was made.

Immunotherapy with 3 days of 1 g intravenous methylprednisolone followed by oral prednisolone (initial dose 1 mg/kg daily) gradually improved her visual field defects, although the BNH remained (Fig. 1G). Additionally, CFF ameliorated bilaterally at 18.7 Hz and 29.2 Hz in the right eye and left eye, respectively. OCT showed improvement in macular thickness in both eyes. Although the pretreatment information was not available, the decrease in peripapillary retinal nerve fiber layer thickness (RNFL) in both eyes was found especially in the temporal sectors (Fig. 1H), consistent with the BNH. Gadolinium enhancement MRI of the bilateral optic nerve sheaths showed a reduction in signal intensities after treatment (Fig. 1I).

2. Discussion

The underlying causes of BNH can be classified into three categories according to the anatomical location, (i) ocular lesions, such as keratoconus, glaucoma, drusen, and retinitis pigmentosa, (ii) optic nerve lesions including optic chiasm, such as syphilitic ON, ischemic optic neuropathy, internal carotid artery aneurysm and atherosclerosis, pituitary apoplexy, pneumosinus dilatans, and olfactory groove meningioma, (iii) intracranial lesions, such as brain tumor, the elevation of intracranial pressure, and occipital lobe lesions [6,7]. Salinas-Garcia et al. reported in 75% of cases with BNH originate from ocular lesions or optic nerve [8]. Our case showed lack of ocular and intracranial...

**Abbreviation:** BNH, binasal hemianopia; CFF, central critical flicker frequency; GVF, Goldmann visual fields; MRI, magnetic resonance imaging; OCT, ocular coherence tomography; ON, optic neuritis; OPN, optic perineuritis; RNFL, retinal nerve fiber layer thickness.
lesions, but RAPD in the right eye, decrease in CFF in both eyes, and inflammation in both optic nerve sheaths suggesting that optic nerve damage due to OPN was the cause of BNH. Since we lack the pathological analysis of the optic nerve sheath, the actual pathophysiology of visual field defects remains unclear. However, one study with two cases of OPN biopsy revealed the presence of inflammatory cells, perineural fibrosis, vasculitic change, and granuloma in the optic nerve sheaths. The authors presumed that visual loss in these OPN patients could be attributed to secondary ischemic optic neuropathy due to circumferential compression by the thickened optic nerve sheath [2]. Therefore, we also speculate that the unrecovered BNH in our case could be due to irreversible ischemic change in bilateral uncrossed optic nerve fibers caused by chronic inflammation and fibrosis in bilateral optic nerve sheaths.

It is important to distinguish OPN from ON because the underlying diseases and treatment strategies for them are different. There are several distinguishing features between OPN and ON. The average age of onset is more older in patients with OPN than with ON [1,2]. Typically, the central vision is preserved in patients with OPN whereas it is often impaired in ON. Since relapse is more frequent in patients with OPN, more effective treatment is required for a longer duration [2]. Diagnosis of OPN and initiation of treatment in the early stage of the disease course is crucial because treatment delay can lead to irreversible visual impairment [9]. Gadolinium-enhanced fat-saturated T1-weighted MRI is the most useful imaging study for the diagnosis of OPN [10].

In conclusion, this is the first case of BNH due to bilateral OPN, which has been described in the literature. Early diagnosis of OPN is crucial to avoid irreversible visual impairment. Further study is needed to analyze the pattern of visual field defects and the pathological changes of optic nerve sheath in patients with OPN.

**Funding**

This work was supported by the Japan Society for the Promotion of Science (JSPS) KAKENHI (Grant Number 19H03549).

**Ethical publication statement**

The study was approved by the Ethics Committee of the Kumamoto University Hospital. The patient provided written informed consent for

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**Fig. 1.** (A) Goldmann visual fields test (GVF) before treatment: The image shows incomplete binasal hemianopia respecting vertical central line. (B) Funduscopic examination before treatment reveals no swelling or color change of the discs bilaterally. (C) Gadolinium enhancement magnetic resonance imaging: The arrows show bilateral optic nerve sheath enhancement. (D) Gallium scintigraphy: The arrow reveals accumulation in mediastinal lymph nodes. (E-F) Pathology of the lymph node (E) Hematoxylin and eosin (H&E) staining shows non-caseating epithelioid cells granulomas, scale bar = 50 μm. (F) H&E staining shows multinucleated giant cells, scale bar = 20 μm. (G) GVF shows improvement of visual field defects after treatment. (H) Ocular coherence tomography (OCT): “Hotter color” indicating the reduction of bilateral peripapillary retinal nerve fiber layer thickness, especially in the temporal sectors. (I) Gadolinium enhancement magnetic resonance imaging: The arrows show a reduction in signal intensities in bilateral optic nerve sheaths.
the investigations, according to the Declaration of Helsinki.

Declaration of Competing Interest
None.

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