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Case Report

Ocular Blood Flow Changes in a Case with Nonarteritic Ischemic Optic Neuropathy Complicated by Obstructive Sleep Apnea Syndrome

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Abstract

A 49-year-old man with a visual field defect (VFD) in his right eye was transferred to our clinic. He exhibited an enlarged optic nerve head (ONH) cupping and pallor of the ONH rim, despite normal intraocular pressure. Decreased ONH blood flow, compared to the other eye, was detected using laser speckle flowgraphy. He had also been diagnosed with obstructive sleep apnea syndrome. Approximately one and a half years later, his VFD had progressed with further reduction in ONH blood flow. After the patient was administered kallidinogenase tablets for nonarteritic ischemic optic neuropathy, the ONH blood flow was improved and the VFD was ameliorated.

Keywords: *Blood Flow; Kallidinogenase; Laser Speckle Flowgraphy; Nonarteritic Ischemic Optic Neuropathy; Obstructive Sleep Apnea Syndrome; Optic Nerve Head*

Abbreviations

LSFG: Laser Speckle Flowgraphy; MBR: Mean Blur Rate; NAION: Nonarteritic Ischemic Optic Neuropathy; ONH: Optic Nerve Head; OSAS: Obstructive Sleep Apnea Syndrome

Introduction

Nonarteritic ischemic optic neuropathy (NAION) is caused by insufficiency of blood flow through the posterior ciliary artery to the optic nerve [1]. Previously, it has been reported that the optic nerve head (ONH) tissue blood flow, measured with laser speckle flowgraphy (LSFG), of the affected eye was approximately 30% lower than that of the unaffected eye in patients with NAION [2]. The exact cause of NAION is not known; however, systemic conditions accompanying NAION include systemic hypertension, ischemic heart diseases, hypercholesterolemia, atherosclerosis, nocturnal hypotension, and smoking [3-6].

Obstructive sleep apnea syndrome (OSAS) is a common, yet underdiagnosed, condition that may be associated with significant morbidity if left untreated. It is characterized by recurrent interruption of normal breathing during sleep due to upper airway obstruction [7]. OSAS has been associated with many vascular diseases, including cardiovascular disease, hypertension, and stroke [8-10]. It is also suggested that OSAS could create transient hypoxemia and increase vascular resistance, which may compromise ONH perfusion [11]. It has been stressed that OSAS may be a predisposing condition for ischemic optic neuropathy [12].

In the current report, we present a case of NAION complicated by OSAS, with reduced ONH blood flow detected using LSFG.

Case Presentation

A 49-year-old man had been prescribed contact lenses for myopia at a clinic. He underwent a visual field test because enlargement of his ONH cupping and nerve fiber layer defect (NFLD) in his right eye (OD) was pointed out (Figures 1 and 2). Consequently, he was found to have visual field defect (VFD) in OD (Figure 3). There were no abnormal findings in his left eye (OS). His refractive error, best-corrected visual acuity, and intraocular pressure were -2.50 D, 24/20, and 10 mmHg, respectively, in both eyes.

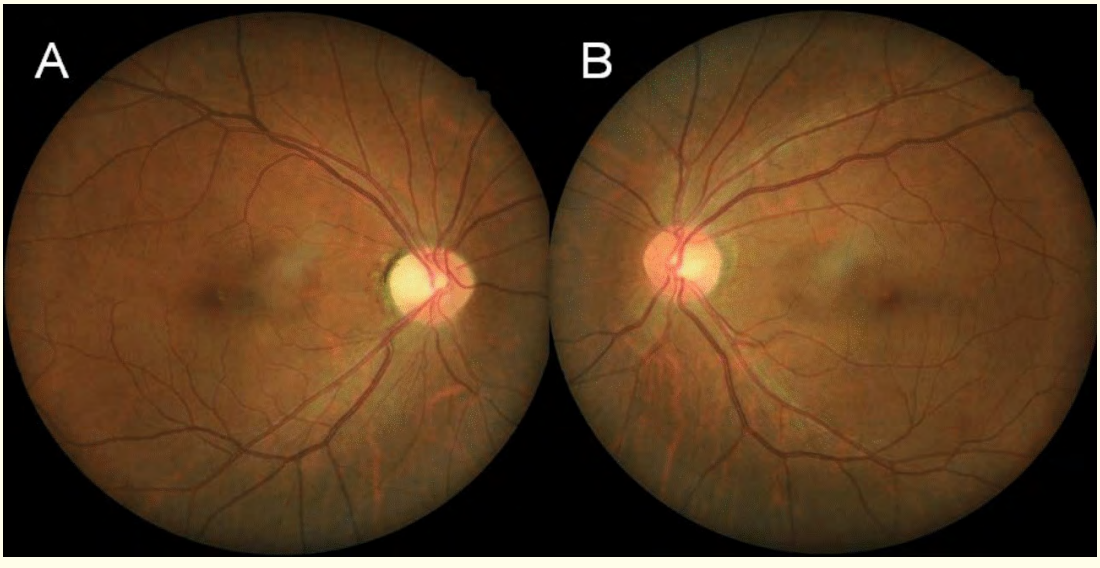

Figure 1: *Fundus photographs taken at the patient's visit to the previous clinic (A: right eye, B: left eye) in April 2014.*

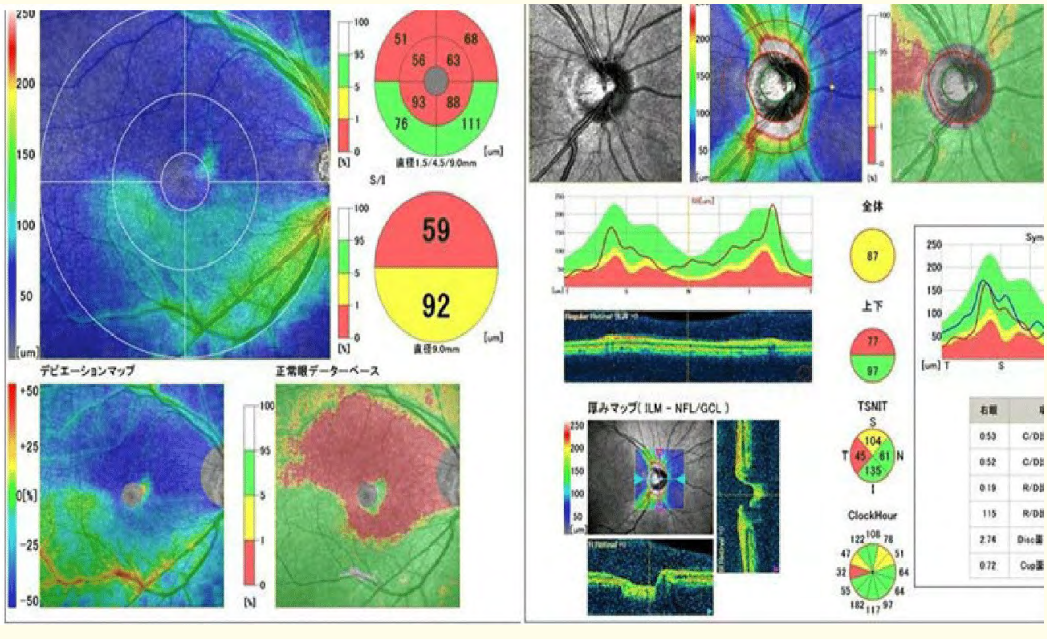

Figure 2: *Optical coherence tomography findings from the patient's right eye at the same visit as in figure 1.*

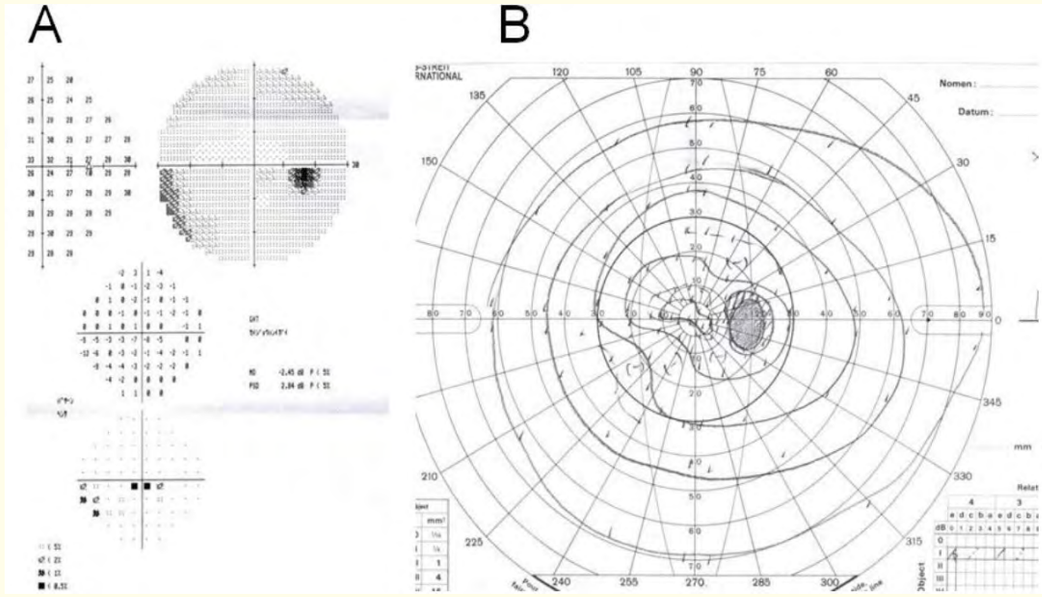

Figure 3: *Visual field findings from the patient's right eye, obtained using the Humphrey field analyzer (A) and with the Goldmann perimeter (B) a few days after the same visit as shown in figure 1.*

The doctor (ophthalmologist) at the clinic suspected that this patient had glaucoma and transferred him to our clinic. Measurement of ONH blood flow on LSFG-NAVI (Softcare Co., Ltd., Fukuoka, Japan) indicated reduced blood flow in the rim and cupping in OD (Figure 4). Although his VFD was consistent with glaucoma, the pallor and reduced blood flow in his ONH rim seemed incompatible with glaucomatous changes. In addition, he had been diagnosed with OSAS for which he had been treated for a few years in another clinic. We decided to observe him without any treatment for a while because he suffered little subjective symptoms at that stage.

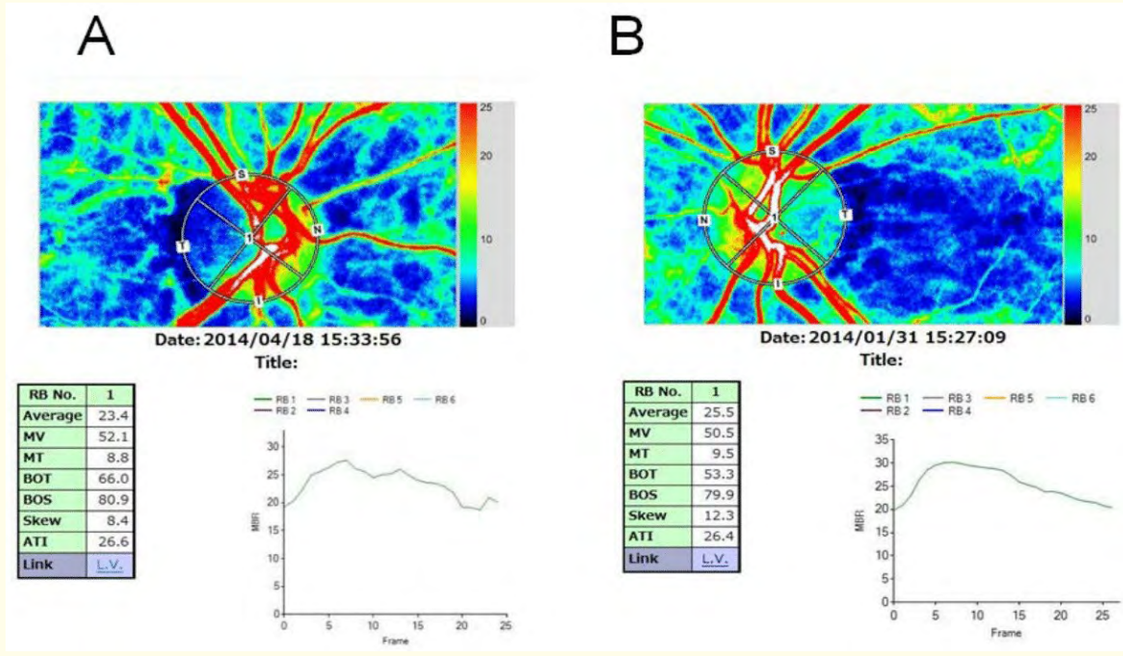

Figure 4: *Findings of optic nerve head blood flow, obtained using laser speckle flowgraphy at the first visit to our clinic in April 2014 (A: right eye, B: left eye).*

Approximately one and a half years after his first visit to our clinic, he suffered a slightly blurred vision in OD. At this time, the visual field tests indicated that his blind spot of Mariotte had spread to the fixating point (Figure 5). His best-corrected visual acuity had deteriorated to 18/20 in OD. The tissue component of the ONH blood flow (MT) and blowout time in OD had further reduced to 8.2 and 53.4, respectively, as compared to the first measurement (8.8 and 66.0, respectively). MT changes in both eyes during the observation period are shown in figure 6. His intraocular pressure was around 10 mmHg throughout the observation period.

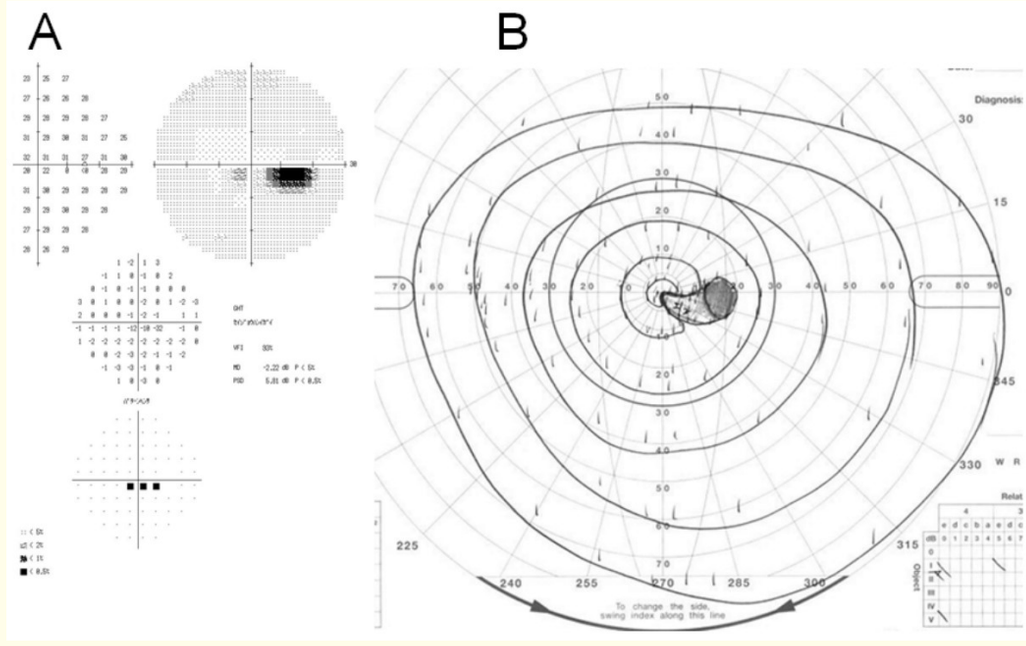

Figure 5: *Visual field findings from the patient's right eye, obtained using the Humphrey field analyzer (A) and with the Goldmann perimeter (B) in November 2015.*

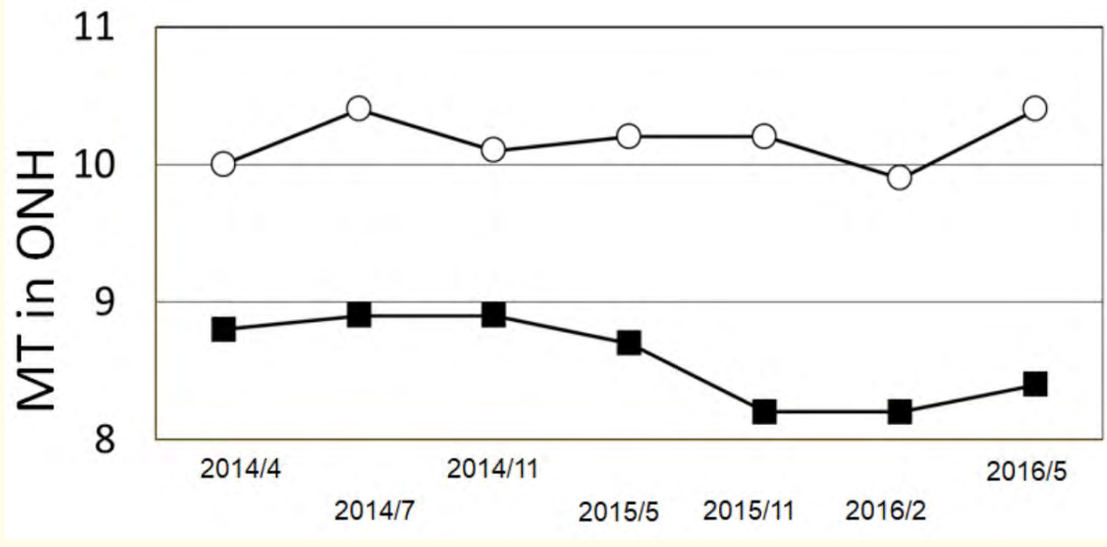

Figure 6: *Changes in the tissue component of optic nerve head blood flow (MT) throughout the observation period (closed squares: right eye, open circles: left eye).*

We started prescribing kallidinogenase tablets (Sanwa Kagaku Kenkyusho Co., Ltd., Nagoya, Japan) for him under diagnosis of NAION, because he had no systemic arteritis and was relatively young (49 years). His VFD in OD gradually ameliorated 3 and 6 months after starting prescription of kallidinogenase tablets (Figure 7). His best-corrected visual acuity recovered to 20/20 in OD. In addition, 6 months later, MT and blowout time of the ONH blood flow in OD had increased to 8.4 and 58.6, respectively.

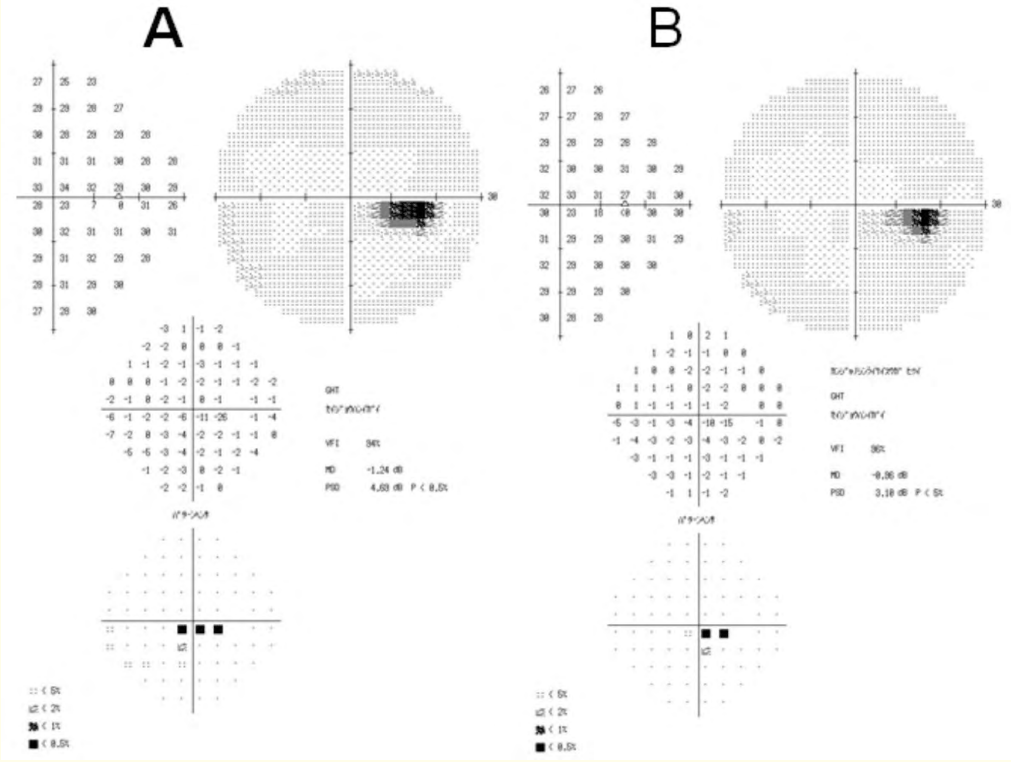

Figure 7: *Visual field findings from the patient's right eye, obtained using the Humphrey field analyzer, 3 and 6 months after starting treatment with kallidinogenase tablets (A: 3 months, B: 6 months).*

The principles of methods for determining ONH blood flow using LSFG have been previously described [13]. Mydriasis was induced with 1 - 2 drops of topical 0.5% tropicamide (Mydrin-M Ophthalmic Solution; Santen Pharmaceutical Co., Ltd., Osaka, Japan) for measurement of ONH blood flow. ONH blood flow was measured in a sitting position. Mean blur rate (MBR) was used as an indicator of blood flow [14]. MT, the MBR of the tissue component of the ONH, was automatically calculated using the LSFG Analyzer software (Ver. 7.0.26.0, Softcare, Ltd.). Blowout time is an indicator that represents the time that the wave maintains more than half of the mean of maximum and minimum MBR during a heartbeat [14]. A high blowout time indicates that blood flow is maintained at a high level for a long time during each heartbeat, ensuring that the peripheral tissue is supplied with sufficient blood.

Discussion

This report describes the time-course of changes in ONH blood flow, detected using LSFG, in a patient with NAION complicated by OSAS. Previous reports have suggested a relationship between OSAS and NAION. The prevalence of OSAS in patients with NAION was reported to be 75% to 95% [15-17]. Furthermore, the prevalence of OSAS was higher in patients with NAION than in a control group [18].

Although these results suggest an association between NAION and OSAS, to the best of our knowledge, there have been no reports regarding ocular blood flow changes in a patient with NAION as well as OSAS to date. LSFG was helpful for diagnosing NAION and evaluating the effects of therapy.

At the patient's first visit to our clinic, his symptoms were similar to those of glaucoma, since he had VFD, enlargement and cupping of his ONH, and NFLD in one of his eyes. However, the pallor and reduced blood flow in the ONH rim suggested that he suffered from NAION. Since his symptoms were rather mild and atypical, we first observed him without treatment, although the treatment for OSAS was continued in another clinic. When he suffered slightly blurred vision at one and a half years after his first visit to our clinic, his VFD had progressed further and visual acuity was reduced. Moreover, LSFG indicated further reduced ONH blood flow. These findings suggested that he had suffered the second or later attack of NAION at this time. Since his VFD had progressed to the extent that the fixating point was affected, we commenced treatment.

His VFD was ameliorated and ONH blood flow was improved after starting kallidinogenase tablets, suggesting that this medication could induce improvement of ONH blood flow and visual function in a patient with NAION. Previously we reported that the same medication was effective in a patient with normal-tension glaucoma [19].

In that patient, the enhanced short-term fluctuation was reduced and VFD was improved after starting the medication. Although these two patients may not have the same pathological condition, both conditions are probably related to disturbance of ONH blood flow. There are several reports indicating the effects of kallidinogenase in animals with ischemic retinal and ONH damage [20,21] and in a patient with central retinal vein occlusion [22]. Therefore, oral kallidinogenase tablets may be a candidate treatment for NAION. The effectiveness of this treatment is worth investigating in clinical tests in future.

Conclusion

We presented a case of NAION complicated by OSAS. This report demonstrated that LSFG was useful for diagnosing NAION and for evaluating the effect of therapy.

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Conflict of Interest

The authors state that the manuscript has not been published previously, and they have no conflict of interest.

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