American Journal of Ophthalmology Case Reports 38 (2025) 102316

Contents lists available at ScienceDirect

American Journal of Ophthalmology Case Reports

journal homepage: www.ajocasereports.com/

Evaluation of blood flow in arteritic anterior ischemic optic neuropathy using laser speckle flowgraphy: A case series

Chiaki Yamaguchi [a], Naoki Kiyota [a], Noriko Himori [a,b], Takahiro Oshima [a], Takayuki Takeshita [c], Kazuko Omodaka [a], Satoru Tsuda [a], Toru Nakazawa [a,d,e,f,*]

[a] Department of Ophthalmology, Tohoku University Graduate School of Medicine, Sendai, Miyagi, Japan
[b] Department of Aging Vision Healthcare, Tohoku University Graduate School of Biomedical Engineering, Sendai, Miyagi, Japan
[c] Department of Ophthalmology, Osaki City Hospital, Osaki, Miyagi, Japan
[d] Department of Ophthalmic Imaging and Information Analytics, Tohoku University Graduate School of Medicine, Sendai, Miyagi, Japan
[e] Department of Retinal Disease Control, Tohoku University Graduate School of Medicine, Sendai, Miyagi, Japan
[f] Department of Advanced Ophthalmic Medicine, Tohoku University Graduate School of Medicine, Sendai, Miyagi, Japan

ARTICLE INFO

Keywords:
Arteritic anterior ischemic optic neuropathy
Laser speckle flowgraphy
Ocular blood flow
Steroid therapy
Case series

ABSTRACT

Background: Arteritic anterior ischemic optic neuropathy (AAION), primarily caused by giant cell arteritis, is a leading cause of blindness. This disease results in significant ocular blood flow (BF) impairment, though data on ocular hemodynamics are limited.
Methods: This observational case series enrolled four patients treated for AAION (age: 81.8 ± 7.8 years; male to female ratio: 3:1) who underwent laser speckle flowgraphy (LSFG) scanning at the initial visit and after steroid treatment in both eyes. Mean blur rate (MBR), an LSFG parameter that represents BF velocity, was obtained in the optic nerve head vessel area (ONH-MV), ONH tissue area (ONH-MT), and peripapillary choroid, in addition to common ophthalmologic parameters.
Results: At the initial visit, all affected eyes had no light perception in best-corrected visual acuity (BCVA) testing, and three cases had a severe increase in circumpapillary retinal nerve fiber layer thickness (cpRNFLT; 191.00 ± 42.03 μm). Pre-treatment, all affected eyes showed decreases in ONH-MV, ONH-MT, and choroidal MBR; this improved after steroid treatment by 80.3 ± 107.6 %, 39.1 ± 79.7 %, and 289.4 ± 303.4 %, respectively. Pre-treatment, all fellow eyes showed no impairment in BCVA or changes in the cpRNFLT. Post-treatment, two fellow eyes showed an increase in ONH-MV and ONH-MT parameters (72.8 ± 32.6 % and 82.2 ± 22.3 %, respectively), while all fellow eyes showed an increase in choroidal MBR (152.7 ± 126.1 %).
Conclusion: LSFG could be useful for monitoring ocular BF changes in eyes with AAION and asymptomatic fellow eyes before and after steroid treatment.

1. Claim of priority statement

After conducting a literature review on March 5, 2025, utilizing PubMed, Google Scholar, and other relevant databases, using the keywords: "arteritic anterior ischemic optic neuropathy," "laser speckle flowgraphy," and "steroid treatment," we did not find any prior reports of this specific combination in the literature.

2. Introduction

Arteritic anterior ischemic optic neuropathy (AAION) is a leading cause of blindness.[1,2] AAION is associated with giant cell arteritis (GCA), which leads to vasculitis and severe blood flow (BF) impairment.[1] Additionally, AAION develops in the unaffected eye in about 50 % of cases within days to weeks, highlighting the need for accurate diagnosis and disease monitoring.[3]

Visual acuity (VA) and visual field testing are essential in diagnosing and following AAION patients, but they are subjective.[1,2] Optical coherence tomography (OCT) is objective but does not capture ocular BF. OCT angiography can visualize the vasculature and has been reported to effectively diagnose AAION.[4] However, due to AAION's rarity, we are not aware of studies that have investigated hemodynamics in

* Corresponding author. Department of Ophthalmology, Tohoku University Graduate School of Medicine, 1-1 Seiryo-machi, Aoba-Ku, Sendai, Miyagi, 980-8574, Japan.
E-mail address: toru.nakazawa.e1@tohoku.ac.jp (T. Nakazawa).

https://doi.org/10.1016/j.ajoc.2025.102316
Received 11 September 2024; Received in revised form 5 March 2025; Accepted 6 March 2025
Available online 24 March 2025

AAION (i.e., BF reduction at onset and the response to steroid treatment).

Laser speckle flowgraphy (LSFG) is a non-invasive, reproducible method for measuring ocular BF; images are captured in just 4 s without any dye.[5] We reported that LSFG is useful not only for measuring optic nerve head (ONH) BF and peripapillary choroidal BF (ppCBF) to diagnose optic neuropathies[6] but also for monitoring ocular BF during glaucoma follow-up.[7,8]

This case series presents ocular BF data from four AAION patients examined with LSFG before and after steroid treatment.

3. Subjects and methods

This retrospective, observational case series was observed at Tohoku University Hospital in Sendai, Japan, and the study was approved by the institutional review board of Tohoku University Graduate School of Medicine. All procedures were approved by the ethics committee of Tohoku University Graduate School of Medicine and followed the Declaration of Helsinki and its later amendments or comparable ethical standards. This case series is reported in line with the PROCESS guideline.[9]

This case series comprised four Japanese patients treated for AAION who underwent LSFG examinations before and after steroid treatment from March 2013 to April 2024. AAION was diagnosed by a neuro-ophthalmology expert (N.H.) and was defined as anterior ischemic optic neuropathy with GCA; GCA was diagnosed based on the 2022 ACR/EULAR classification criteria for GCA, which require a score of $\geq$6 points. The criteria include an absolute requirement of age $\geq$50 years at the time of diagnosis. Additional clinical criteria include morning stiffness in the shoulders/neck (+2), sudden visual loss (+3), jaw or tongue claudication (+2), new temporal headache (+2), scalp tenderness (+2), and abnormal findings in the temporal artery (+2). Laboratory, imaging, and biopsy criteria comprise the following: a maximum erythrocyte sedimentation rate (ESR) of $\geq$50 mm/h or a maximum C-reactive protein (CRP) level of $\geq$1.0 mg/dL (+3), positive findings from a temporal artery biopsy (TAB), or the presence of a halo sign in temporal artery ultrasounds (+5) (the criteria for TAB findings include definitive vasculitis characterized by mononuclear leukocyte infiltration and fragmentation of the internal elastic lamina), bilateral axillary involvement (+2), and FDG-PET activity throughout the aorta (+2).[10]

Decimal best-corrected VA (BCVA) was converted to logarithmic minimum angle of resolution (logMAR) for analysis. Intraocular pressure was measured with non-contact tonometry (Tonoref III; Nidek Co., Ltd, Gamagori, Japan). Circumpapillary retinal nerve fiber layer thickness (cpRNFLT) was determined with the 3D OCT-2000 and its embedded software (ver. 8.11, Topcon Corporation, Tokyo, Japan). ONH and choroidal BF were assessed with the LSFG-NAVI device (Softcare Co., Ltd., Fukutsu, Japan), which measures mean blur rate (MBR) in arbitrary units (AU). MBR represents relative BF velocity, which is calculated automatically by the LSFG software (LSFG Analyzer, version 3.1.59.0) based on the contrast of the speckle pattern generated by laser light scattered by moving red blood cells.[11] First, an ellipsoid band was manually drawn around the ONH to define the region of interest (ROI) in composite MBR color maps. The LSFG software then automatically divided the large-vessel and tissue (i.e., capillary) areas of the ONH and determined vessel-area MBR (MV) and tissue-area MBR (MT) separately in a cross-sectional analysis. ONH-MT has been reported to be a good indicator of BF in the deep capillaries of the ONH.[12] A same-sized ROI was then set on the temporal side of the ONH in the same image to mark the peripapillary choroid region, as we described previously.[13] Before the LSFG measurements, the patients pupils were dilated with 0.4 % tropicamide, a muscarinic antagonist (Mydrin M; Santen Pharmaceutical Co., Ltd.). After instillation, the patients sat in a dark, quiet room for 15 minutes to stabilize pupil dilation, blood pressure (BP), and pulse rate (PR). Then, BP and PR were measured (HBP-1300; Omron Colin Co., Ltd. Tokyo, Japan) to evaluate the effects of systemic hemodynamics, and the LSFG examination was performed.

For the statistical analysis, means $\pm$ standard deviation and percentages were used in descriptive statistics for clinical features and ophthalmological findings. The lower limits of MBR were calculated as follows:

$$\text{Lower limit of the normal range of MBR} = \text{Mean value of MBR in the control} - 1.96 \times \text{SD of MBR in the control}$$

The significance level for comparisons between AAION-affected eyes and controls was $P < 0.05$. All statistical analyses were performed with R (version 4.3.0; https://www.R-project.org/).

4. Results

The pre-treatment clinical backgrounds of the AAION patients are shown in Table S1. Ophthalmologic findings of AAION patients pre- and post-steroid treeatment are shown in Table S2. The average age was 81.8 $\pm$ 7.8 years, with 3 males and 1 female. Their medical histories included hypertension (HT) in 3 patients and diabetes mellitus (DM) in 1 patient. ESR was higher than calculated normal values based on age and sex (43.38 $\pm$ 4.88 vs. 80.25 $\pm$ 29.44 mm/h).[14] Compared to reference values for the elderly, the CRP level was higher (0.54 $\pm$ 1.04 vs. 3.26 $\pm$ 2.21 mg/dl).[15] Pre-treatment, all affected eyes showed no light perception (NLP). **Case 1**, **case 2,** and **case 3** showed increased cpRNFLT (191.00 $\pm$ 42.03 μm). No fellow eyes showed any obvious VA loss (0.12 $\pm$ 0.13 logMAR) or increase in cpRNFLT (107.50 $\pm$ 19.02 μm). The LSFG color map of the affected eyes showed many cold-colored areas (Figs. 1–4), suggesting a decrease in ocular BF. We quantified this through comparison with LSFG data from eight randomly selected age- and sex-matched individuals from our medical records. All affected eyes showed a decrease in ONH-MV (34.65 $\pm$ 4.63 vs. 17.58 $\pm$ 10.86 AU, $P < 0.001$), ONH-MT (10.28 $\pm$ 2.17 vs. 5.47 $\pm$ 3.42 AU, $P = 0.002$), and choroidal MBR (7.42 $\pm$ 1.50 vs. 2.63 $\pm$ 1.80 AU, $P = 0.001$) (Supplementary Fig. S1). In the three cases of fluorescein angiography (FA) performed at our institution, watershed zones were observed in the early phase in all three cases, and delayed BF filling in the central retinal artery (CRA) was observed in two cases (Supplementary Fig. S2).

Post-treatment, the affected eyes in **case 2** and **case 3** showed slightly improved visual function (from NLP to counting fingers and light perception, respectively). All affected eyes demonstrated a decrease in cpRNFLT (from 166.56 $\pm$ 60.48 to 86.0 $\pm$ 29.7 μm). **Case 2**, **case 3**, and **case 4** showed improved ONH-MV and ONH-MT, and all cases showed improved choroidal MBR (Fig. 5; **upper panel**). The average increase in all affected eyes was 80.3 $\pm$ 107.6 % for ONH-MV, 39.1 $\pm$ 79.7 % for ONH-MT, and 289.4 $\pm$ 303.4 % for choroidal MBR.

The lower limit of normal values calculated from the control group was 25.6 AU for ONH-MV, 6.0 AU for ONH-MT, and 4.5 AU for choroidal MBR. Fig. 5, lower panel, illustrates the changes in MBR in the fellow eyes in response to steroid treatment. In **case 2**, ONH-MV increased from 14.2 AU to 29.2 AU, ONH-MT increased from 3.7 AU to 7.6 AU, and choroidal MBR increased from 1.6 AU to 5.6 AU, all exceeding the respective lower limits for normal values, indicating normalization. In **case 3**, ONH-MV increased from 25.5 AU to 35.8 AU, ONH-MT increased from 5.7 AU to 9.0 AU, and choroidal MBR increased from 1.3 AU to 5.1 AU, all exceeding the respective lower limits for normal values, indicating normalization. In **case 1** and **case 4**, ONH-MV and ONH-MT did not show significant increases and remained stable within the normal range. Choroidal MBR also remained within the normal range both pre- and post-treatment but demonstrated further improvement following treatment. The average increase in all fellow eyes was 33.8 $\pm$ 49.1 % for ONH-MV, 39.6 $\pm$ 53.2 % for ONH-MT, and 152.7 $\pm$ 126.1 % for choroidal MBR.

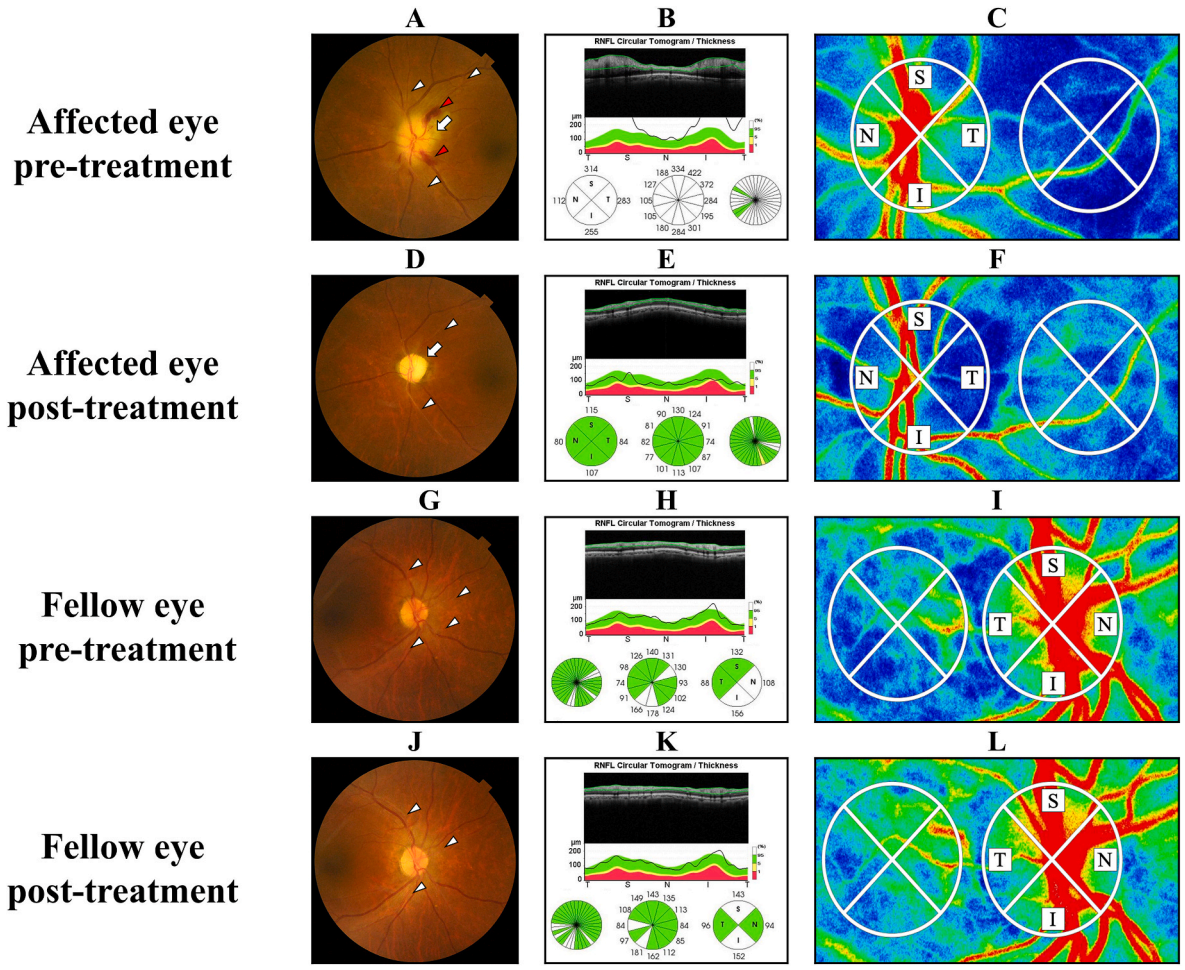

Fig. 1. Case 1. (A)–(C) show the pre-treatment affected eye (the left eye), (D) to (F) show the post-treatment affected eye (the left eye), (G) to (I) show the pre-treatment fellow eye (the right eye), and (J) to (L) show the post-treatment fellow eye (the right eye).
(A) Optic nerve head (ONH) swelling (white arrow), papillary hemorrhage (red arrowhead), and retinal artery narrowing (white arrowhead) are evident in this fundus photograph.
(B) Circumpapillary retinal nerve fiber layer thickness (cpRNFLT) is 241.0 μm in this optical coherence tomography (OCT) image.
(C) Laser speckle flowgraphy (LSFG) measurements of ONH vessel-area mean blur rate (MBR; ONH-MV) (30.7 AU), ONH tissue-area MBR (ONH-MT) (8.6 AU), and choroidal MBR (4.2 AU).
(D) A pale ONH (white arrow) and retinal artery narrowing (white arrowhead) can be observed in this fundus photograph.
(E) CpRNFLT has decreased to 96.0 μm.
(F) LSFG measurements for ONH-MV (26.6 AU), ONH-MT (4.9 AU), and choroidal MBR (6.1 AU).
(G) The retinal arteries are narrow (white arrowhead) in this fundus photograph.
(H) CpRNFLT is 121.0 μm.
(I) ONH-MV is 45.1 AU, ONH-MT is 12.0 AU, and choroidal MBR is 6.3 AU.
(J) Findings from fundus photography, including narrow retinal arteries (white arrowhead), remain unchanged.
(K) CpRNFLT is 121.0 μm.
(L) ONH-MV is 44.2 AU, ONH-MT is 11.9 AU, and choroidal MBR is 7.6 AU.

5. Case presentation

5.1. Case 1

A 79-year-old woman with HT and polymyalgia rheumatica (PMR) suddenly complained of blurred vision in her left eye ten days before admission. The patient was taking 10 mg of systemic steroids for the treatment of PMR. The ESR was 78.0 mm/h, and CRP was 3.2 mg/dl. The patient was negative for headache, and there were no positive findings from a TAB. The patient scored 6 points based on the ACR/EULAR classification criteria for GCA. Considering the presence of PMR, which is known to be associated with GCA,[16] and subsequent steroid responsiveness, this case was definitively diagnosed as AAION and qualified to be included in this study. The patient was treated with intravenous methylprednisolone (1000 mg/day) for three days, followed by prednisone (30 mg daily, tapered monthly).

In the affected eye (the left eye), pre-treatment BCVA was NLP, and a relative afferent pupillary defect (RAPD) was observed. ONH swelling with papillary hemorrhage was observed in a fundus photograph (Fig. 1A), and cpRNFLT was 241.0 μm (Fig. 1B). ONH-MV was 30.7 AU, ONH-MT was 8.6 AU, and choroidal MBR was 4.2 AU (Fig. 1C). In FA, a watershed zone was observed in the superior, nasal, inferior, and parts of the peripapillary regions of the ONH (Supplementary Fig. S2B). After one month of treatment, BCVA remained NLP, and the RAPD was unchanged. A pale ONH was observed in a fundus photograph (Fig. 1D), and cpRNFLT decreased to 96.0 μm (Fig. 1E). ONH-MV and ONH-MT decreased, and choroidal MBR increased (Figs. 1F and 5).

In the fellow eye (the right eye), pre-treatment BCVA was 20/30. The

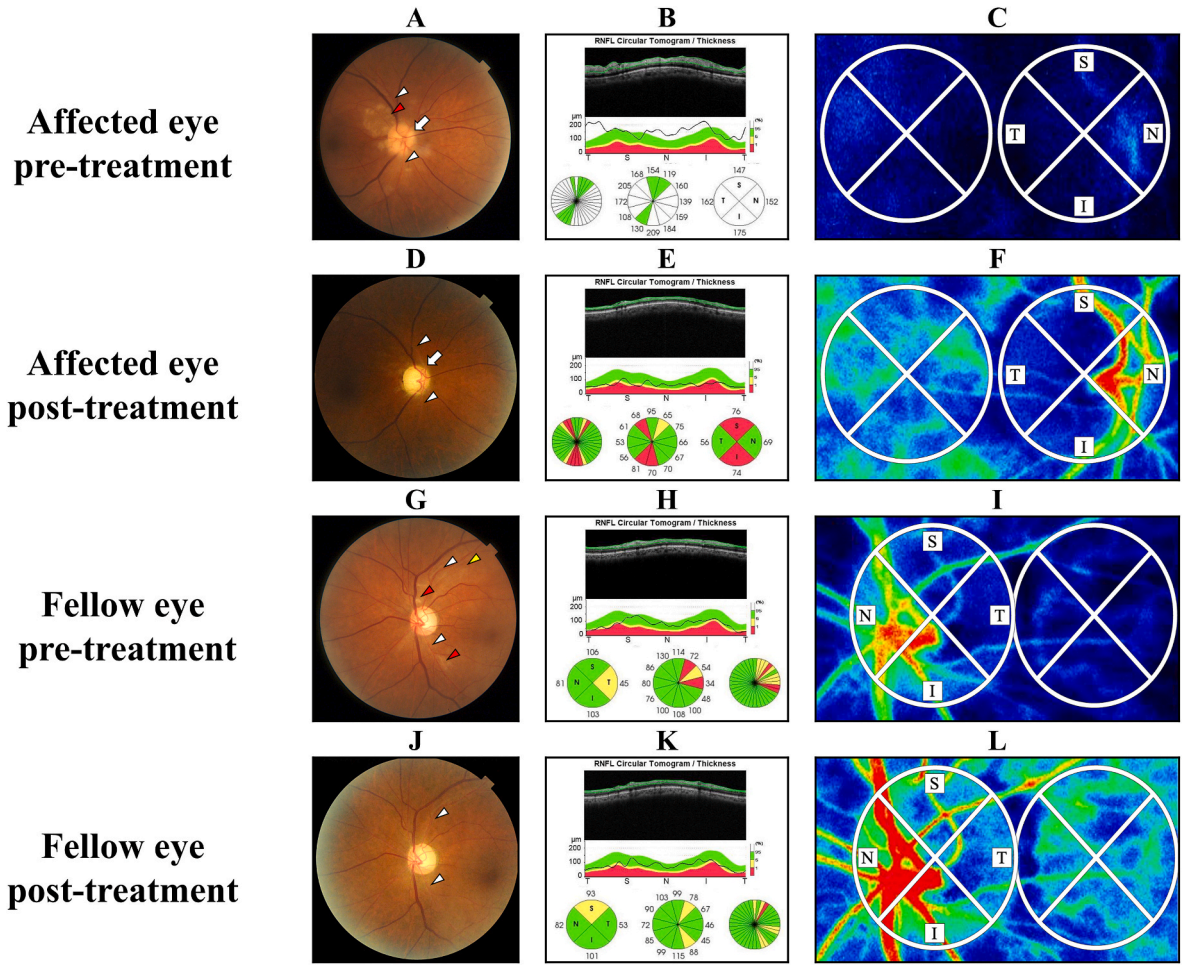

Fig. 2. Case 2. (A)–(C) show the pre-treatment affected eye (the right eye), (D) to (F) show the post-treatment affected eye (the right eye), (G) to (I) show the pre-treatment fellow eye (the left eye), and (J) to (L) show the post-treatment fellow eye (the left eye).
(A) ONH swelling (white arrow), soft exudates (red arrowhead), and retinal artery narrowing (white arrowhead) are evident in this fundus photograph.
(B) CpRNFLT is 159.0 μm.
(C) ONH-MV is 4.4 AU, ONH-MT is 1.5 AU, and choroidal MBR is 1.6 AU.
(D) A pale ONH (white arrowhead) and retinal artery narrowing (white arrowhead) can be observed in this fundus photograph.
(E) CpRNFLT has decreased to 69.0 μm.
(F) ONH-MV is 15.9 AU, ONH-MT is 3.7 AU, and choroidal MBR is 6.4 AU.
(G) In this fundus photograph, retinal artery narrowing (white arrowhead), soft exudates (red arrowhead), and retinal nerve fiber defects are evident in the superior region (yellow arrowhead).
(H) CpRNFLT is 83.0 μm.
(I) ONH-MV is 14.2 AU, ONH-MT is 3.7 AU, and choroidal MBR is 1.6 AU.
(J) Findings from fundus photography: narrow retinal arteries (white arrowhead) remain unchanged; however, soft exudates have improved.
(K) CpRNFLT is 82.0 μm.
(L) ONH-MV is 29.2 AU, ONH-MT is 7.6 AU, and choroidal MBR is 5.6 AU.

retinal arteries were narrow in a fundus photograph (Fig. 1G), and cpRNFLT was 121.0 μm (Fig. 1H). ONH-MV was 45.1 AU, ONH-MT was 12.0 AU, and choroidal MBR was 6.3 AU (Fig. 1I). FA revealed no significant findings (Supplementary Fig. S2A). After the treatment, BCVA was 20/16. The findings from fundus photography remained unchanged (Fig. 1J), and cpRNFLT was unchanged at 121.0 μm (Fig. 1K). The changes in ONH-MV and ONH-MT were not significant, whereas choroidal MBR increased (Figs. 1L and 5).

5.2. Case 2

A 73-year-old male with no medical history suddenly complained of blurred vision in his right eye ten days before admission. ESR was 120.0 mm/h, and CRP was 1.9 mg/dl. There were positive results from a TAB, and the patient scored 11 points based on the ACR/EULAR classification criteria for GCA, leading to a diagnosis of GCA. The patient was treated with intravenous methylprednisolone (1000 mg/day) for three days, followed by prednisone (50 mg daily, tapered monthly).

In the affected eye (the right eye), pre-treatment BCVA was NLP, and a RAPD was observed. ONH swelling and soft exudates were observed in a fundus photograph (Fig. 2A), and cpRNFLT was 159.0 μm (Fig. 2B). ONH-MV, ONH-MT, and choroidal MBR were low at 4.4 AU, 1.5 AU, and 1.6 AU, respectively (Fig. 2C). In FA, delayed BF filling in the CRA and a watershed zone extending from the temporal to the inferior regions of the ONH were observed (Supplementary Fig. S2C). After two months of treatment, BCVA remained NLP, and the RAPD was unchanged. A pale ONH was observed in the fundus photograph (Fig. 2D), and cpRNFLT decreased to 69.0 μm (Fig. 2E). ONH-MV, ONH-MT, and choroidal MBR increased (Figs. 2F and 5).

In the fellow eye (the left eye), pre-treatment BCVA was 20/22. In a

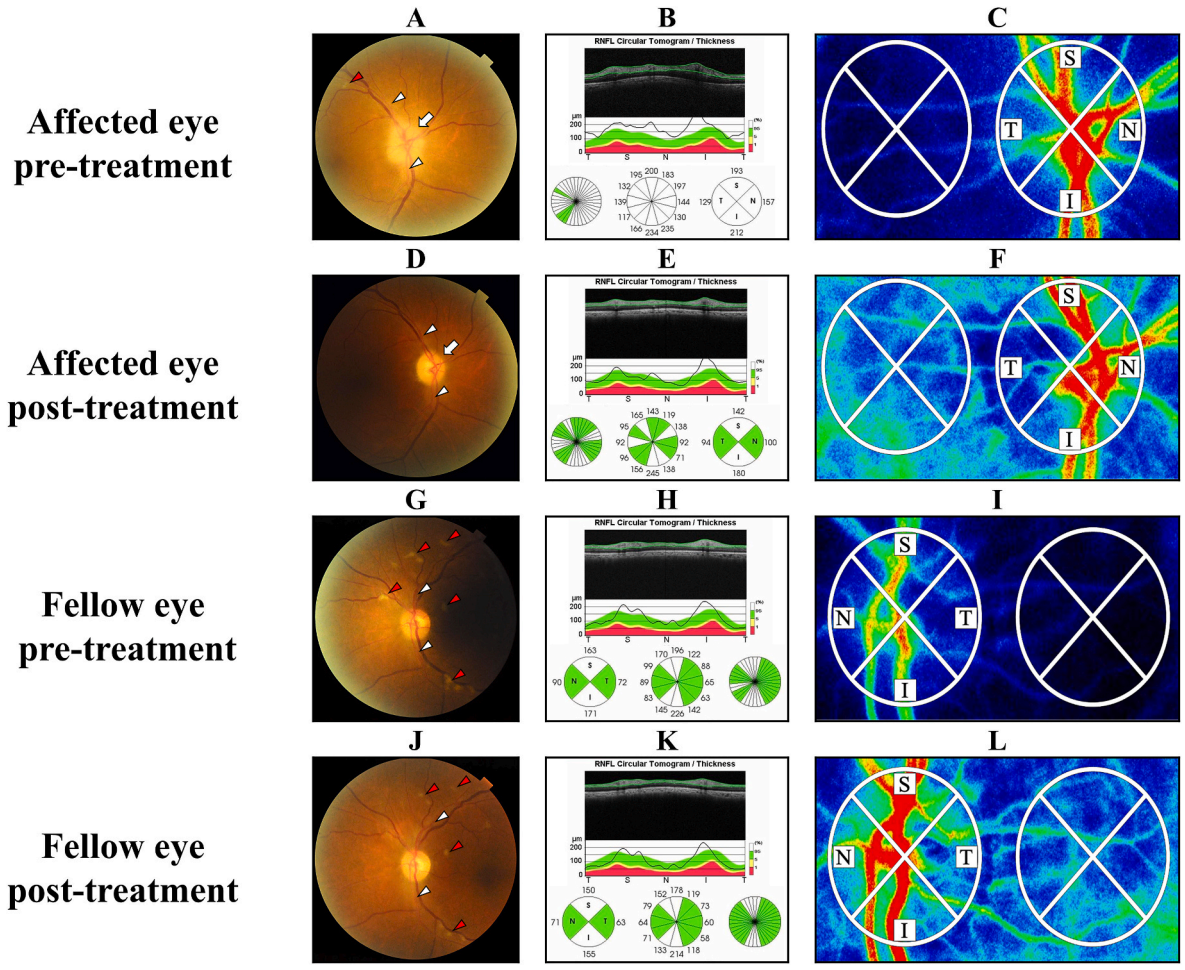

Fig. 3. Case 3. (A)–(C) show the pre-treatment affected eye (the right eye), (D) to (F) show the post-treatment affected eye (the right eye), (G) to (I) show the pre-treatment fellow eye (the left eye), and (J) to (L) show the post-treatment fellow eye (the left eye).
(A) ONH swelling (white arrow), soft exudates (red arrowhead), and retinal artery narrowing (white arrowhead) are evident in this fundus photograph.
(B) CpRNFLT is 173.0 μm.
(C) ONH-MV is 15.6 AU, ONH-MT is 3.8 AU, and choroidal MBR is 0.6 AU.
(D) Mild ONH swelling (white arrow) and retinal artery narrowing (white arrowhead) are evident in this fundus photograph; however, soft exudates have improved.
(E) CpRNFLT has decreased to 129.0 μm.
(F) ONH-MV is 20.4 AU, ONH-MT is 4.8 AU, and choroidal MBR is 5.1 AU.
(G) This fundus photograph shows retinal artery narrowing (white arrowhead) and soft exudates (red arrowhead).
(H) CpRNFLT is 124.0 μm.
(I) ONH-MV is 25.5 AU, ONH-MT is 5.7 AU, and choroidal MBR is 1.3 AU.
(J) This fundus photograph shows retinal artery narrowing (white arrowhead) and improvement in the soft exudates (red arrowhead).
(K) CpRNFLT is 94.0 μm.
(L) ONH-MV is 35.8 AU, ONH-MT is 9.0 AU, and choroidal MBR is 5.1 AU.

fundus photograph, the retinal artery was narrowed. Retinal nerve fiber defects were evident in the superior region (Fig. 2G), and cpRNFLT was 83.0 μm (Fig. 2H). ONH-MV, ONH-MT, and choroidal MBR were low, at 14.2 AU, 3.7 AU, and 1.6 AU, respectively (Fig. 2I). FA revealed no significant findings (Supplementary Fig. S2D). After the treatment, BCVA was 20/16. The findings from fundus photography remained unchanged (Fig. 2J), and cpRNFLT was 82.0 μm (Fig. 2K). ONH-MV, ONH-MT, and choroidal MBR increased (Figs. 2L and 5).

5.3. Case 3

An 83-year-old male with HT and DM suddenly complained of blurred vision in his right eye four days before admission. He had a new onset of localized headache and temporal artery tenderness. ESR was 49.0 mm/h, and CRP was 6.4 mg/dl. The patient did not consent to a TAB but scored 8 points based on the ACR/EULAR classification criteria for GCA, leading to a diagnosis of GCA. The patient was treated with intravenous methylprednisolone (1000 mg/day) for three days, followed by prednisone (30 mg daily, tapered monthly).

In the affected eye (the right eye), pre-treatment BCVA was NLP, and a RAPD was observed. ONH swelling was observed in the fundus photograph (Fig. 3A), and cpRNFLT was 173.0 μm (Fig. 3B). ONH-MV, ONH-MT, and choroidal MBR were low at 15.6 AU, 3.8 AU, and 0.6 AU, respectively (Fig. 3C). FA was not performed at our institution. After four months of treatment, BCVA remained NLP, and the RAPD was unchanged. Mild ONH swelling was observed in fundus photography (Fig. 3D), and cpRNFLT decreased to 129.0 μm (Fig. 3E). ONH-MV, ONH-MT, and choroidal MBR increased (Figs. 3F and 5).

In the fellow eye (the left eye), pre-treatment BCVA was 20/22. In the fundus photograph, there was a narrowing of the retinal arteries and soft exudates (Fig. 3G), and cpRNFLT was 124.0 μm (Fig. 3H). ONH-MV, ONH-MT, and choroidal MBR were low, at 25.5 AU, 5.7 AU, and 1.3 AU,

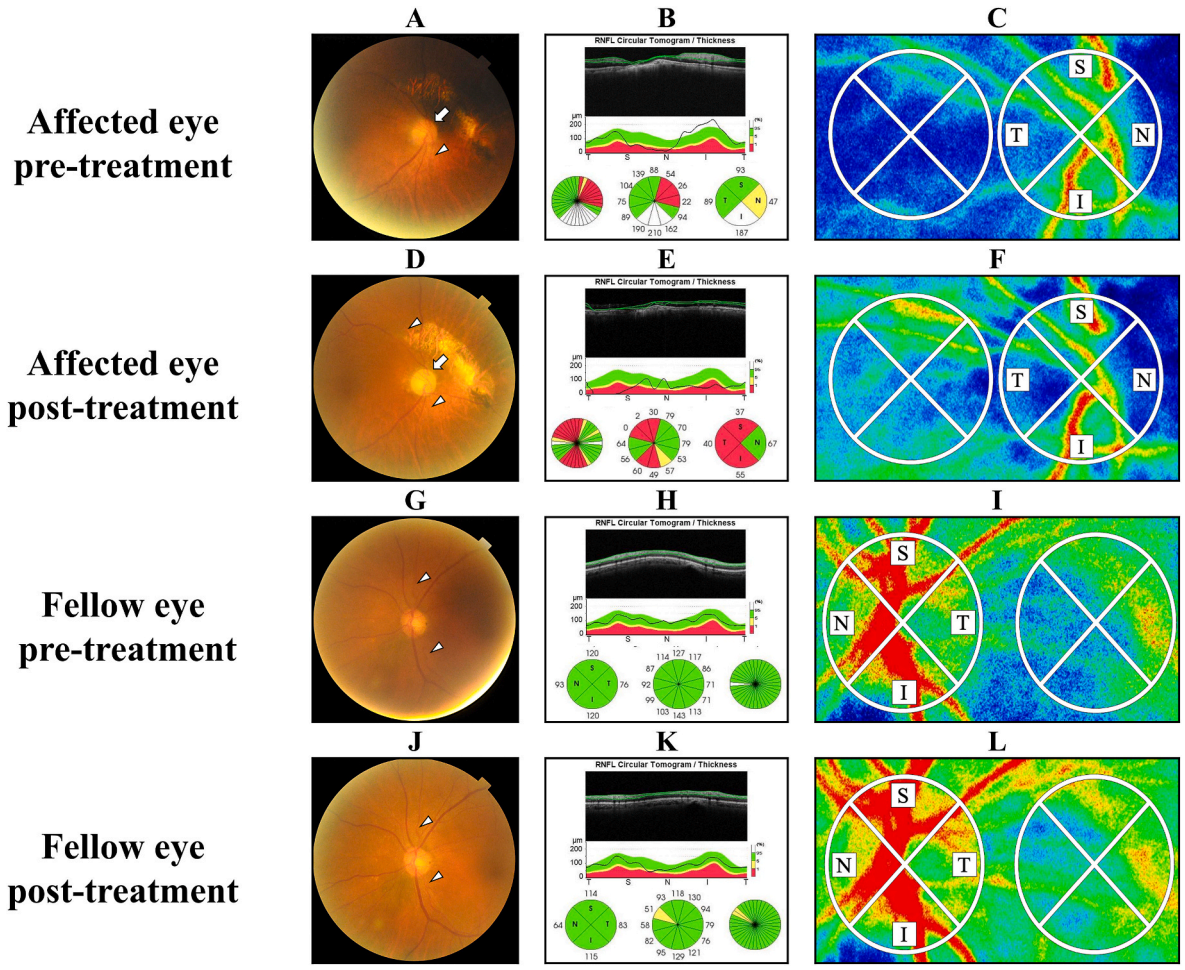

Fig. 4. Case 4. (A)–(C) show the pre-treatment affected eye (the right eye), (D) to (F) show the post-treatment affected eye (the right eye), (G) to (I) show the pre-treatment fellow eye (the left eye), and (J) to (L) show the post-treatment fellow eye (the left eye).
(A) A reddish ONH (white arrow) and retinal artery narrowing (white arrowhead) are evident in this fundus photograph.
(B) CpRNFLT is 104.0 μm.
(C) ONH-MV is 19.7 AU, ONH-MT is 8.0 AU, and choroidal MBR is 4.1 AU.
(D) A pale ONH (white arrow) and retinal artery narrowing (white arrowhead) are evident in this fundus photograph.
(E) CpRNFLT has decreased to 50.0 μm.
(F) ONH-MV is 27.6 AU, ONH-MT is 9.4 AU, and choroidal MBR is 6.7 AU.
(G) There is narrowing of the retinal arteries (white arrowhead) in this fundus photograph.
(H) CpRNFLT is 102.0 μm.
(I) ONH-MV is 38.4 AU, ONH-MT is 14.4 AU, and choroidal MBR is 8.2 AU.
(J) Findings from fundus photography, including narrow retinal arteries (white arrowhead), remain unchanged.
(K) CpRNFLT is 94.0 μm.
(L) ONH-MV is 35.2 AU, ONH-MT is 13.6 AU, and choroidal MBR is 12.3 AU.

respectively (Fig. 3I). After the treatment, BCVA improved to 20/16. Findings from fundus photography showed improvement in the soft exudates (Fig. 3J), and cpRNFLT decreased to 94.0 μm (Fig. 3K). ONH-MV, ONH-MT, and choroidal MBR increased (Figs. 3L and 5).

5.4. Case 4

A 92-year-old man with HT and PMR suddenly complained of blurred vision in his right eye eight days before admission. The patient was taking 15 mg of systemic steroids for the treatment of PMR. ESR was 74.0 mm/h, and CRP was 1.5 mg/dl. There were positive results from a TAB, and the patient scored 11 points based on the ACR/EULAR classification criteria for GCA, leading to a diagnosis of GCA. The patient was treated with intravenous methylprednisolone (1000 mg/day) for three days, followed by prednisone (30 mg daily, tapered monthly).

In the affected eye (the right eye), pre-treatment BCVA was NLP, and a RAPD was observed. A reddish ONH was observed in fundus photography (Fig. 4A), and cpRNFLT was 104.0 μm (Fig. 4B). ONH-MV, ONH-MT, and choroidal MBR were low, at 19.7 AU, 8.0 AU, and 4.1 AU, respectively (Fig. 4C). In FA, delayed BF filling in the CRA was observed, with a watershed zone extending from the nasal to the inferior regions of the ONH. Additionally, window defects were identified in a chorioretinal atrophy area surrounding the ONH (Supplementary Fig. S2E). After two months of treatment, BCVA remained NLP, and the RAPD was unchanged. A pale ONH was observed in fundus photography (Fig. 4D), and cpRNFLT decreased to 50.0 μm (Fig. 4E). ONH-MV, ONH-MT, and choroidal MBR increased (Figs. 4F and 5).

In the fellow eye (the left eye), pre-treatment BCVA was 20/33. The fundus photograph was normal (Fig. 4G), and cpRNFLT was 102.0 μm (Fig. 4H). ONH-MV was 38.4 AU, ONH-MT was 14.4 AU, and choroidal MBR was 8.2 AU (Fig. 4I). FA revealed no significant findings (Supplementary Fig. S2F). After the treatment, BCVA improved to 20/

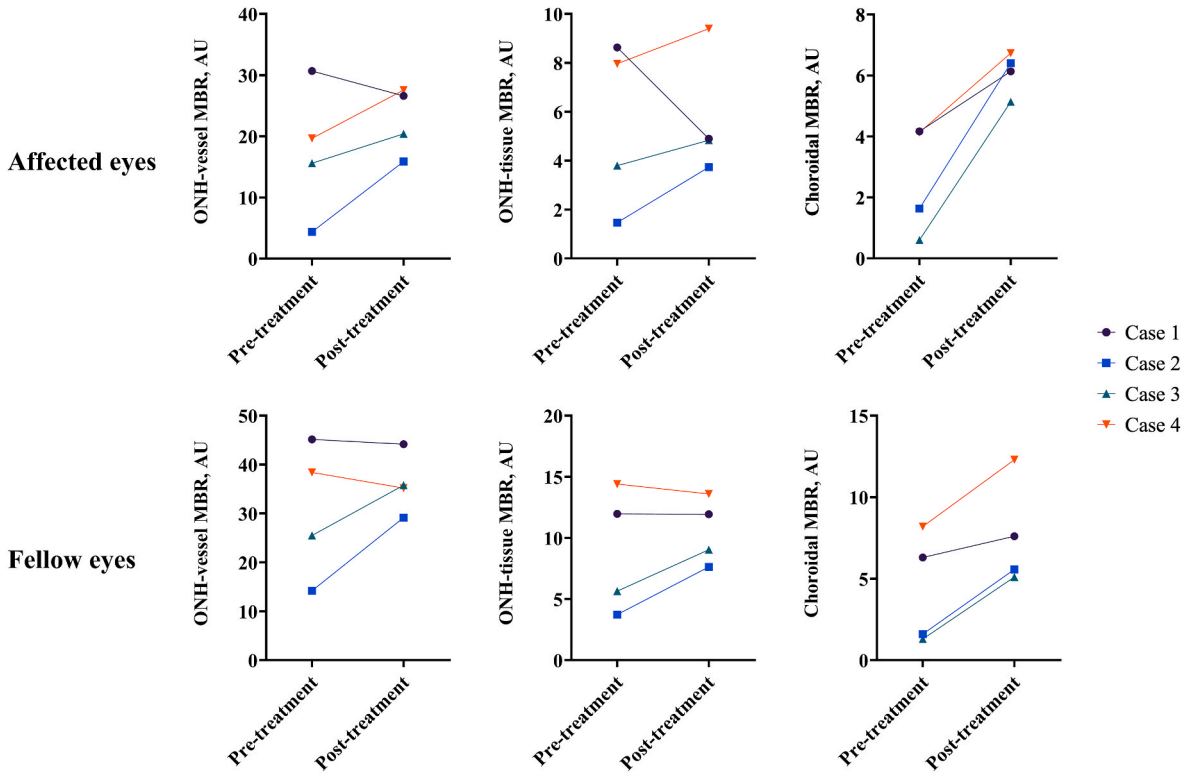

Fig. 5. Changes in mean blur rate (MBR), measured with laser speckle flowgraphy, are presented pre-and post-treatment with systemic steroids. The upper panel shows affected eyes, and the lower panel shows fellow eyes. From left to right: optic nerve head vessel-area MBR (ONH-MV), ONH tissue-area MBR (ONH-MT), and choroidal MBR. The black lines represent **case 1**, the blue lines represent **case 2**, the green lines represent **case 3**, and the orange lines represent **case 4**.

30. Findings from fundus photography remained normal (Fig. 4J), and cpRNFLT was 94.0 μm (Fig. 4K). ONH-MV and ONH-MT showed no statistically significant changes, while choroidal MBR increased (Figs. 4L and 5).

6. Discussion

In this study, we evaluated BF before and after systemic steroid treatment to understand the dynamics of BF in AAION patients. Pre-treatment, all affected eyes showed severe BF impairment in the ONH large vessels, ONH tissue, and peripapillary choroid. Post-treatment, three affected eyes showed an improvement in the ONH large vessels and tissue BF, and all affected eyes showed an improvement in the ppCBF. Post-treatment, two fellow eyes showed increased ONH large-vessel and tissue BF. Additionally, all fellow eyes showed increased ppCBF.

In this study, all AAION-affected eyes showed a significant decrease in ONH large-vessel and ONH tissue BF, and ppCBF compared to age- and sex-matched controls. MBR in the large-vessel area reflects BF from the CRAs.[17] In contrast, MBR in the tissue and choroidal areas reflects BF from the short posterior ciliary arteries (SPCAs).[17] In this study, the delayed BF filling of the CRA in FA was also consistent with decreased ONH-MV, and the watershed zone, associated with impairment of the SPCAs, was consistent with decreased ONH-MT and choroidal MBR, which supports the findings of previous studies. The etiology of GCA involves vasculitis, which causes thickening of the vessel walls in the CRAs, SPCAs, and upstream vessels, thereby increasing vascular resistance.[18] Thus, it is reasonable that MBR decreased in all three areas in this study.

Three AAION-affected eyes showed an improvement in ONH large-vessel and tissue BF, and all affected eyes showed an improvement in ppCBF. Our results indicated that ocular BF increased through the treatment's anti-inflammatory effects on vasculitis. While steroids themselves have a BF-improving effect,[19,20] considering the significant decrease in BF at the onset, the increase in ocular BF observed in this study can mainly be attributed to the therapeutic effect of the treatment on vasculitis.

This study showed reduced ONH large-vessel BF, ONH tissue BF, and ppCBF in two fellow eyes. The reduction in ocular BF in fellow eyes is consistent with the reduction in vessel density within the ONH reported by Pierro L et al.[21] These reductions in BF suggest that the CRAs and SPCAs are also impaired in the fellow eyes of patients with AAION when it occurs, even when FA does not reveal BF impairment. Therefore, understanding the severity of BF impairment in the fellow eye using LSFG can provide insights into the extent of vasculitis and potentially help assess the risk of fellow-eye involvement.

In this study, two fellow eyes with impaired BF showed increased BF in the ONH large-vessel and tissue areas, and all fellow eyes exhibited increased ppCBF. Importantly, the two cases in which BF impairment was observed before treatment showed normalization of BF parameters after treatment. Our results suggest that this may be related to the improvement of vasculitis in the CRAs and SPCAs due to steroids. Therefore, even if fellow eyes are asymptomatic, LSFG may be useful for monitoring the improvement of vasculitis due to steroids.

In conclusion, LSFG can noninvasively and efficiently evaluate the hemodynamics of AAION-affected and fellow eyes at onset and before and after steroid treatment. The objective measurement of LSFG parameters may play a supplementary role in diagnosing AAION, determining treatment efficacy, and assessing the severity of fellow-eye involvement.

CRediT authorship contribution statement

Chiaki Yamaguchi: Writing – original draft, Visualization, Project administration, Methodology, Investigation, Formal analysis, Data curation, Conceptualization. **Naoki Kiyota:** Writing – original draft,

Visualization, Project administration, Methodology, Conceptualization. **Noriko Himori:** Writing – original draft, Visualization, Validation, Supervision, Project administration, Methodology, Conceptualization. **Takahiro Oshima:** Methodology, Data curation, Conceptualization. **Takayuki Takeshita:** Writing – review & editing, Validation, Conceptualization. **Kazuko Omodaka:** Validation, Supervision, Methodology, Conceptualization. **Satoru Tsuda:** Validation, Project administration, Methodology, Conceptualization. **Toru Nakazawa:** Writing – review & editing, Validation, Supervision, Project administration, Methodology, Investigation, Conceptualization.

Patient consent

Consent to publish the case reports was not obtained. This report does not contain any personal information that could lead to the identification of the patient.

Funding

Financial support for this research was provided in part by the Japan Society for the Promotion of Science (JSPS) KAKENHI Grants-in-Aid for Scientific Research (B) (grant number: 17H04349; recipient: T.N.) and grants from the Center of Innovation Program (COI-NEXT) by the Japan Science and Technology Agency (grant number: JPMJPF2201). The funders had no role in the design or conduct of the study; collection, management, analysis, or interpretation of the data; preparation, review, or approval of the manuscript; or the decision to submit the manuscript for publication.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Acknowledgment

The authors thank Mr. Tim Hilts for reviewing and editing the manuscript's language. We thank Kenji Okamoto of Softcare Co., Ltd. for his valuable comments on the manuscript.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.ajoc.2025.102316.

References

1. Hayreh SS. Ischemic optic neuropathy. *Prog Retin Eye Res*. 2009;28(1):34–62.
2. Bajpai V, Madan S, Beri S. Arteritic anterior ischaemic optic neuropathy: an update. *Eur J Ophthalmol*. 2021;31(6):2818–2827.
3. Arnold AC. Ischemic optic neuropathies. *Ophthalmol Clin North Am*. 2001;14(1):83–98.
4. Balducci N, Morara M, Veronese C, et al. Optical coherence tomography angiography in acute arteritic and non-arteritic anterior ischemic optic neuropathy. *Graefe's Arch Clin Exp Ophthalmol*. 2017;255(11):2255–2261.
5. Sugiyama T, Araie M, Riva CE, Schmetterer L, Orgul S. Use of laser speckle flowgraphy in ocular blood flow research. *Acta Ophthalmol*. 2010;88(7):723–729.
6. Yamaguchi C, Kiyota N, Himori N, Omodaka K, Tsuda S, Nakazawa T. Differentiating optic neuropathies using laser speckle flowgraphy: evaluating blood flow patterns in the optic nerve head and peripapillary choroid. *Acta Ophthalmol*. 2024;103:e49–e57.
7. Kiyota N, Shiga Y, Ninomiya T, et al. The effect of β-blocker eye drops on pulse rate, ocular blood flow, and glaucoma progression: a retrospective longitudinal study. *Adv Ther*. 2024;41(2):730–743.
8. Kiyota N, Shiga Y, Omodaka K, Pak K, Nakazawa T. Time-course changes in optic nerve head blood flow and retinal nerve fiber layer thickness in eyes with open-angle glaucoma. *Ophthalmology*. 2021;128(5):663–671.
9. Mathew G, Sohrabi C, Franchi T, et al. Preferred reporting of case series in surgery (PROCESS) 2023 guidelines. *Int J Surg*. 2023;109(12):3760–3769.
10. Ponte C, Grayson PC, Robson JC, et al. American College of Rheumatology/EULAR classification criteria for giant cell arteritis. *Ann Rheum Dis*. 2022;81(12):1647–1653.
11. Sugiyama T. Basic Technology and clinical applications of the updated model of laser speckle flowgraphy to ocular diseases. *Photonics*. 2014;1(3):220–234.
12. Aizawa N, Nitta F, Kunikata H, et al. Laser speckle and hydrogen gas clearance measurements of optic nerve circulation in albino and pigmented rabbits with or without optic disc atrophy. *Investig Ophthalmol Vis Sci*. 2014;55(12):7991–7996.
13. Kiyota N, Shiga Y, Omodaka K, Nakazawa T. The relationship between choroidal blood flow and glaucoma progression in a Japanese study population. *Jpn J Ophthalmol*. 2022;66(5):425–433.
14. Miller A, Green M, Robinson D. Simple rule for calculating normal erythrocyte sedimentation rate. *Br Med J*. 1983;286(6361):266.
15. Woloshin S, Schwartz LM. Distribution of C-reactive protein values in the United States. *N Engl J Med*. 2005;352(15):1611–1613.
16. Buttgereit F, Dejaco C, Matteson EL, Dasgupta B. Polymyalgia rheumatica and giant cell arteritis: a systematic review. *JAMA*. 2016;315(22):2442–2458.
17. Wang L, Cull GA, Piper C, Burgoyne CF, Fortune B. Anterior and posterior optic nerve head blood flow in nonhuman primate experimental glaucoma model measured by laser speckle imaging technique and microsphere method. *Investigative Opthalmology & Visual Science*. 2012;53(13):8303–8309.
18. Samson M, Corbera-Bellalta M, Audia S, et al. Recent advances in our understanding of giant cell arteritis pathogenesis. *Autoimmun Rev*. 2017;16(8):833–844.
19. Cabañas F, Pellicer A, García-Alix A, Quero J, Stiris TA. Effect of dexamethasone therapy on cerebral and ocular blood flow velocity in premature infants studied by colour Doppler flow imaging. *Eur J Pediatr*. 1997;156(1):41–46.
20. Lew H, Lee SY, Jang JW, Kim HY, Kang SJ, Kim SJ. The effects of high-dose corticosteroid therapy on optic nerve head blood flow in experimental traumatic optic neuropathy. *Ophthalmic Res*. 1999;31(6):463–470.
21. Pierro L, Arrigo A, Aragona E, Cavalleri M, Bandello F. Vessel density and vessel tortuosity quantitative analysis of arteritic and non-arteritic anterior ischemic optic neuropathies: an optical coherence tomography angiography study. *J Clin Med*. 2020;9(4):1094.