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Glaucoma-Like Compressive Optic Neuropathy Associated With a Supraclinoid Internal Carotid Artery Aneurysm: A Case Report

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Abstract

Compressive optic neuropathy can mimic glaucoma, and the classic clues to compression may be absent or subtle. An 80-year-old woman reported that she had been undergoing fundus examinations for diabetes mellitus and follow-up of a right retinal nerve fiber layer (RNFL) defect at another clinic. Over several days, she noticed a vague difficulty seeing with the right eye, without headache, ocular pain, or diplopia, and was referred to our hospital by a different clinic. Decimal visual acuity was 0.7 in the right eye and 0.9 in the left eye, and intraocular pressure (IOP) was 18 mm Hg in both eyes. A mild right relative afferent pupillary defect (RAPD) was present. The right optic disc appeared essentially normal, with a cup-to-disc ratio of 0.3 and no rim thinning or pallor. Optical coherence tomography (OCT) showed a localized inferotemporal RNFL defect, but the average peripapillary RNFL thickness was within normal limits. In contrast, macular ganglion cell-inner plexiform layer (GCIPL) thickness was reduced in both the superior and inferior hemispheres. Humphrey 30-2 perimetry showed glaucoma-like loss, predominantly nasal and involving the superior and inferior hemifields, with a mean deviation of −16.45 dB. This field loss was out of proportion to the disc appearance and the RNFL findings. The optic disc, OCT, and visual field findings in the left eye were normal. The new symptom and the RAPD at normal IOP prompted magnetic resonance imaging (MRI) and magnetic resonance angiography (MRA). Imaging showed compression of the right optic nerve by an approximately 10-mm supraclinoid internal carotid artery aneurysm. The aneurysm was treated with a flow diverter at another hospital, and postoperative visual acuity and visual field data were not available. Comparison with six previously reported cases shows that aneurysmal compression can present with glaucoma-like findings at normal IOP and that visual outcomes after treatment are variable. In eyes with a glaucoma-like RNFL defect or visual field loss, three features should raise the possibility of a nonglaucomatous cause, including aneurysmal compression: unilateral damage at normal IOP, a recent change in vision, and field loss out of proportion to the disc and RNFL findings. These features lower the threshold for neuroimaging.

Categories: Neurology, Ophthalmology
Keywords: compressive optic neuropathy, glaucoma mimic, intracranial aneurysm, normal-tension glaucoma, rnfl defect

Introduction

Glaucomatous optic neuropathy is characterized by optic disc cupping, retinal nerve fiber layer (RNFL) defects, and corresponding visual field loss. These findings are not specific to glaucoma. Compressive optic neuropathy can produce the same RNFL thinning, arcuate or nasal visual field defects, and in some cases optic disc cupping [1]. When intraocular pressure (IOP) is within the normal range, the two conditions are difficult to distinguish. Such damage may then be attributed to normal-tension glaucoma (NTG), glaucomatous optic neuropathy without elevated IOP [2]. NTG is partly a diagnosis of exclusion, because other optic neuropathies must be ruled out [3].

The supraclinoid segment of the internal carotid artery lies inferolateral to the optic nerve and lateral to the optic chiasm. Depending on their origin and direction of projection, aneurysms arising from this segment may compress the optic nerve or the chiasm [4] and may present with progressive visual dysfunction [5,6]. Unlike most other glaucoma mimics, an unrecognized aneurysm can rupture and cause life-threatening subarachnoid hemorrhage. The risk of rupture varies with aneurysm size, location, and shape [7]. Its recognition therefore matters beyond vision.

Decreased visual acuity, optic disc pallor disproportionate to cupping, and visual field defects that respect the vertical meridian are the classic clues that should prompt neuroimaging [3,8]. These clues may be absent or subtle. We describe a patient with a localized RNFL defect and glaucoma-like visual field loss at normal IOP, whose visual acuity was only mildly reduced and whose optic disc appeared essentially normal. In this patient, neuroimaging was prompted by a new subjective change in vision, accompanied by a relative afferent pupillary defect (RAPD). We compare the case with six previously reported cases to illustrate how aneurysmal compression may be recognized when the classic clues are minimal [9-14]. In the case reported by Nucci et al., reduced visual acuity, dyschromatopsia, and optic disc pallor prompted imaging [12]. In the case reported by Ashok et al., rapid deterioration despite low IOP revealed compression superimposed on

preexisting NTG [14].

Case Presentation

An 80-year-old woman noticed a vague difficulty seeing with the right eye over several days, visited a local clinic, and was referred to our institution. According to the patient, she had been undergoing routine fundus examinations for diabetes mellitus and follow-up of a right RNFL defect at another clinic. The referring clinic had no earlier data. Records from the follow-up clinic, including information on the duration of follow-up, were not available for this report. She had no headache, ocular or retrobulbar pain, diplopia, or vomiting. She had not been treated with antiglaucoma medication, and IOP measured by the referring physician was 19 mm Hg in both eyes.

At the first visit to our institution, decimal visual acuity was 0.7 in the right eye and 0.9 in the left eye, and IOP was 18 mm Hg in both eyes. Mild cataract was present. No conjunctival injection or obvious abnormality in pupil size was noted. A mild right RAPD was detected with the swinging flashlight test; it was not quantified with neutral density filters. Color vision and critical flicker frequency were not tested. The right optic disc appeared essentially normal, with no rim thinning, pallor, notching, or disc hemorrhage. The cup was small, round, and shallow (cup-to-disc ratio 0.3), and fundus photography showed a narrow crescent of peripapillary atrophy temporally. Humphrey 30-2 visual field testing of the right eye was reliable (fixation losses 2/15, false positives 7%, false negatives 11%). The mean deviation (MD) was −16.45 dB ($P < 0.5\%$), and the pattern standard deviation (PSD) was 11.60 dB ($P < 0.5\%$). The visual field index was 53%, the glaucoma hemifield test was outside normal limits, and the foveal threshold was 28 dB. The loss was predominantly nasal, involved the superior and inferior hemifields, and was densest in the inferonasal quadrant, with additional scattered depression in the temporal field (Figures *1A*, *1B*). Fundus photography also suggested a wedge-shaped inferotemporal RNFL defect (Figure *1C*), at the location of the localized defect shown on OCT. No diabetic retinopathy was present. On the same day, spectral-domain optical coherence tomography (OCT) of the right eye showed a localized inferotemporal RNFL defect: 99 µm at 7 o'clock (below the 1st percentile of the age-matched normative database) and 68 µm at 8 o'clock (between the 1st and 5th percentiles). The average peripapillary RNFL thickness (95 µm) and all four quadrants (superior 117 µm, nasal 71 µm, inferior 123 µm, temporal 70 µm) were within normal limits, and the superior clock hours measured 81 to 133 µm (Figure *1D*). Macular ganglion cell-inner plexiform layer (GCIPL) thickness was reduced, with an average of 79 µm (below the 1st percentile). Thickness was 80 µm in the superior hemisphere (between the 1st and 5th percentiles) and 78 µm in the inferior hemisphere (below the 1st percentile). All four inner sectors and both outer inferior sectors were thinned, whereas the outer superior sectors were within normal limits. The thinning involved both the temporal and nasal halves of the macula and did not respect the vertical meridian (Figure *1E*). The optic disc, OCT, and visual field findings in the left eye were normal.

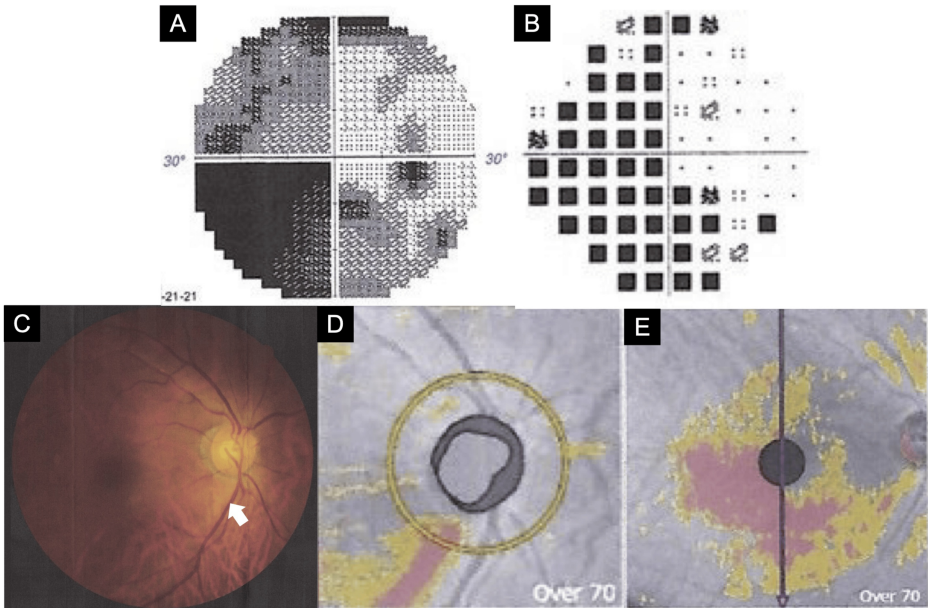


FIGURE 1: Ophthalmic Findings in the Right Eye

Humphrey 30-2 perimetry (grayscale, A; pattern deviation, B) showed glaucoma-like loss, predominantly nasal, involving the superior and inferior hemifields, densest inferonasally, with scattered temporal depression. The mean deviation was −16.45 dB (P < 0.5%), and the pattern standard deviation was 11.60 dB (P < 0.5%). The visual field index was 53%, the foveal threshold was 28 dB, and the glaucoma hemifield test was outside normal limits. Fixation losses were 2/15, false positives 7%, and false negatives 11%. Fundus photography (C) showed an essentially normal optic disc with a small, shallow cup (cup-to-disc ratio 0.3), no rim thinning or pallor, and a narrow temporal crescent of peripapillary atrophy. The arrow marks a suspected inferotemporal retinal nerve fiber layer (RNFL) defect. The peripapillary RNFL deviation map (D) showed a localized inferotemporal defect: 99 µm at 7 o'clock (below the 1st percentile) and 68 µm at 8 o'clock (between the 1st and 5th percentiles). Average RNFL thickness (95 µm) and quadrant thicknesses (superior 117, nasal 71, inferior 123, temporal 70 µm) were within normal limits. The macular ganglion cell–inner plexiform layer deviation map (E) showed thinning in the superior and inferior hemispheres (average 79 µm; superior 80 µm; inferior 78 µm), involving the temporal and nasal halves. Inner-sector thicknesses were 85, 103, 80, and 96 µm in the superotemporal, superonasal, inferotemporal, and inferonasal sectors (the superonasal sector between the 1st and 5th percentiles, the others below the 1st percentile). The corresponding outer-sector values were 65, 93, 65, and 90 µm (the two superior sectors within normal limits, the inferotemporal sector between the 1st and 5th percentiles, and the inferonasal sector below the 1st percentile). Yellow and red indicate thickness between the 1st and 5th percentiles and below the 1st percentile of the age-matched normative database, respectively.

Because of the subjective deterioration over several days and the RAPD at normal IOP, we suspected a nonglaucomatous optic neuropathy and ordered brain magnetic resonance imaging (MRI) with magnetic resonance angiography (MRA) at this visit. Coronal T2-weighted MRI demonstrated compression and deformation of the right optic nerve (Figures *2A*-*2C*), and MRA showed an approximately 10-mm right supraclinoid internal carotid artery aneurysm projecting medially. The patient was transferred to another hospital, where flow-diverter treatment was performed by the neurosurgical team. Postoperative visual acuity and visual field data were not available for this report.

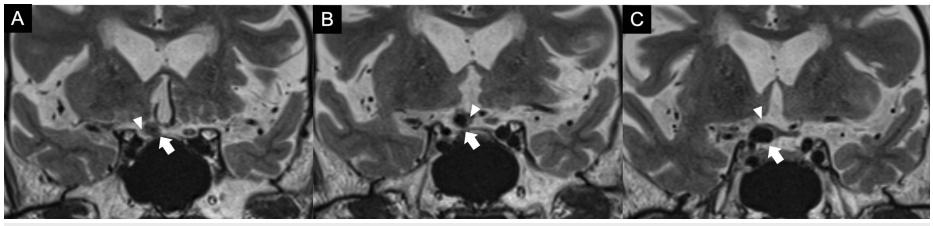


FIGURE 2: Brain Magnetic Resonance Imaging Findings

Consecutive coronal T2-weighted magnetic resonance images showed a lesion suspicious for an approximately 10-mm aneurysm at the right supraclinoid internal carotid artery (A-C, arrows). The lesion projected medially and compressed the right optic nerve (A-C, arrowheads). Magnetic resonance angiography (not shown) confirmed a right supraclinoid internal carotid artery aneurysm, and right compressive optic neuropathy was diagnosed based on the ophthalmic and imaging findings.

Discussion

This patient had a glaucoma-like RNFL defect and visual field loss with IOP in the normal range. The classic clues to compression were absent or subtle: visual acuity was only mildly reduced, the optic disc was not pale, and the visual field pattern resembled glaucoma. The findings that led to neuroimaging were a new subjective change in vision over several days and a mild RAPD. The RAPD must be interpreted with caution. With an MD of −16 dB in one eye and a normal fellow eye, an RAPD is expected whatever the cause. It confirmed asymmetric optic nerve dysfunction but could not, by itself, distinguish compression from asymmetric glaucoma. The new symptom was the more informative clue, because glaucomatous damage at normal IOP usually progresses slowly and without a change that the patient notices over days. Even so, the onset of the structural and visual field abnormalities remained unknown, and the short symptomatic interval should not be taken as evidence that all abnormalities developed acutely.

In retrospect, the findings were also discordant with glaucoma in a way that is recognizable at the first visit. The visual field loss was severe (MD −16.45 dB). Yet the optic disc appeared normal (cup-to-disc ratio 0.3), the average and quadrant RNFL thicknesses were within normal limits, and the only RNFL abnormality was a localized inferotemporal defect. This defect is topographically compatible with the superonasal component of the field loss but not with the dense inferonasal loss, for which the corresponding superotemporal RNFL sectors were preserved. In contrast, macular GCIPL thickness was reduced in both the superior and inferior hemispheres. In glaucoma, visual field loss of this magnitude is usually accompanied by obvious cupping. It is also accompanied by substantial RNFL thinning in the corresponding sectors, allowing for interindividual variability in RNFL thickness [15]. Such a combination is compatible with a recent or ongoing process in which visual field loss and macular GCIPL thinning became detectable while the disc and the average peripapillary RNFL remained within normal limits. Because only one pretreatment assessment was available, however, the actual sequence of these changes could not be established.

Three OCT studies have compared compressive with glaucomatous optic neuropathy [16-18]. The compressive cohorts of Laowanapiban et al. and Lei et al. consisted mainly of sellar or suprasellar mass lesions rather than aneurysmal compression of a single optic nerve, which limits direct extrapolation to our patient [17,18]. In those studies, compression was characterized by thinner nasal and temporal RNFL sectors [16], relative preservation of the inferior RNFL quadrant [18], and nasal hemimacular GCIPL thinning [17]. Glaucoma was characterized by larger cups [16] and inferotemporal GCIPL thinning [17]. Our patient did not show these compressive patterns: the nasal and temporal RNFL quadrants were normal, the only RNFL abnormality was a localized inferotemporal defect, and GCIPL thinning involved both halves of the macula and included the inferotemporal sector. The OCT distribution therefore provided no specific pattern of compression and was, if anything, glaucoma-like. Laowanapiban et al. reported disproportionately mild OCT thinning relative to visual field loss in a subset of newly diagnosed compressive eyes [17]. Our patient's structure-function mismatch resembled this pattern. Visual field loss with preserved RNFL thickness may partly reflect dysfunction of axons that have not yet been lost, and visual field recovery after decompression has been reported in compressive optic neuropathy [17]. These observations argue for prompt evaluation when compression is suspected.

For clinical context, we compared this patient with six previously reported cases of carotid-ophthalmic, ophthalmic segment, or supraclinoid internal carotid artery aneurysms with glaucoma-like findings, identified by a focused PubMed/MEDLINE search with reference tracking and Google Scholar citation searching (final search date May 2, 2026) [9-14]. Their clinical features, treatments, and reported visual outcomes are summarized with those of our patient in Table *1*. Reported outcomes varied. Visual acuity and visual fields were stable or improved after endovascular treatment in two patients [10,12], visual acuity declined to 20/200 during observation in one [11], and diffuse visual field sensitivity loss was reported 3 months after treatment in another [13]. In the patient with preexisting NTG, visual acuity improved after aneurysm closure, but RNFL thinning progressed, and the visual field possibly worsened [14]. The patient of Portney and Roth died of aneurysm rupture three days after clipping [9]. The deficit was therefore not consistently reversible. This comparison provides descriptive context rather than an estimate of frequency or diagnostic accuracy. In the reported cases, the findings that suggested a non-glaucomatous process were visual loss, optic disc pallor, RAPD, dyschromatopsia, rapid progression, and atypical visual field loss, but no single finding was reported in every patient [9-14]. The patient in Nucci et al. had no evident RAPD; reduced visual acuity, dyschromatopsia, and optic disc pallor prompted neuroimaging [12]. In the patient of Ashok et al., rapid deterioration despite low IOP revealed compression superimposed on NTG [14].

Report	Age/Sex	Aneurysm Location	Intraocular Pressure	Glaucoma-like Findings	Findings Suggestive of Compressive Optic Neuropathy	Treatment	Visual Outcome
Present case	80F	Right supraclinoid internal carotid artery,	18/18	Localized inferotemporal RNFL defect; GCIPL thinning; nasal-predominant visual field	Right RAPD; subjective worsening over several days; normal-appearing optic disc (C/D 0.3) and	Flow-diverter treatment	Postoperative ophthalmic examination data not available

		approximately 10 mm		loss with abnormal GHT, HFA 30-2 MD −16.45 dB	average RNFL within normal limits despite severe visual field loss		(treated at another hospital)
Portney et al. [9]	51F	Saccular aneurysm at the left ophthalmic artery-internal carotid artery junction	18/18	Vertically elongated cupping and disc pallor in the left eye. Left inferior hemianopia, superotemporal constriction, and splitting of fixation	RAPD, decreased visual acuity, and pathologic confirmation of cupping caused by prelaminar axon loss and glial column collapse	Clipping	Died 3 days after clipping from aneurysm rupture; no postoperative visual data
Kulkarni et al. [10]	40F	Left internal carotid-ophthalmic artery aneurysm, approximately 14 mm	18/22; during follow-up, 18-24/21-26	Enlarged left C/D ratio. Left superonasal visual field defect and arcuate scotoma	Development of RAPD and unilateral visual field loss not readily explained by anterior uveitis or mild IOP elevation	Coil embolization and internal carotid artery balloon occlusion	Visual acuity and visual field stable after treatment; follow-up duration not stated
Beatty et al. [11]	70F	Left ophthalmic segment, or paraophthalmic, internal carotid artery aneurysm	18/19	Deep left optic disc cupping, enlarged C/D ratio, and rim thinning. Goldmann visual field testing showed a superotemporal arcuate scotoma; HFA 24-2 showed a localized superotemporal defect and abnormal GHT	RAPD and decreased visual acuity	Observation	Left visual acuity declined from 20/40 to 20/200 within 1 month and then remained stable; follow-up visual field not reported
Nucci et al. [12]	41M	Ophthalmic segment internal carotid artery aneurysm crossing the chiasm and predominantly compressing the left optic nerve	16/17	Diagnosis of NTG, asymmetric C/D ratio, and pallor of the residual left neuroretinal rim. Left superonasal relative scotoma and abnormal GHT	Optic disc pallor, dyschromatopsia, decreased visual acuity, and no RAPD	Coil embolization	At 5 months, BCVA improved to 0.0 logMAR in both eyes and visual field defects slightly improved with a residual scotoma
Pincelli Netto et al. [13]	48F	Near the ophthalmic artery origin of the internal carotid artery, 3.5 × 6.5 mm	12/12	Asymmetric C/D ratio and left inferotemporal RNFL defect. Left superior arcuate scotoma and abnormal GHT	Optic disc pallor, dyschromatopsia after 45 days, and headache	Endovascular treatment	At 3 months, headache improved but the visual field showed diffuse loss of sensitivity; postoperative visual acuity not reported
Ashok et al. [14]	72F	Giant left internal carotid-ophthalmic artery aneurysm, 16 mm with an 8-mm neck	8/6	Preexisting NTG, increased left C/D ratio, and OCT RNFL thinning. Progression of left temporal and paracentral visual field loss	Rapid unilateral progression with decreased visual acuity despite low IOP; no RAPD at presentation	Coil embolization after unsuccessful flow diversion and balloon occlusion testing	Left visual acuity improved from light perception to 20/50 after coiling; at 6 weeks, a left RAPD had appeared, visual field testing suggested possible further constriction in the left eye, and OCT showed further RNFL thinning, more marked on the left

TABLE 1: Comparison of the Present Case With Six Previously Reported Cases

Clinical features of the present case and the six previously reported cases identified in the focused literature review are shown. Intraocular pressure is shown in mm Hg as right eye/left eye according to values reported at initial evaluation or around diagnosis. Glaucoma-like findings included optic disc cupping, rim thinning, retinal nerve fiber layer defects, or glaucoma-like visual field abnormalities. Findings suggestive of compressive optic neuropathy included decreased visual acuity, relative afferent pupillary defect, optic disc pallor, dyschromatopsia, rapid progression, or visual field abnormalities atypical for glaucoma. Visual outcome summarizes the visual acuity and visual field findings reported after treatment or during observation, with the follow-up interval when stated. RNFL: retinal nerve fiber layer; GCIPL: ganglion cell-inner plexiform layer; GHT: glaucoma hemifield test; HFA: Humphrey field analyzer; MD: mean deviation; RAPD: relative afferent pupillary defect; C/D: cup-to-disc ratio; IOP: intraocular pressure; NTG: normal-tension glaucoma; BCVA: best-corrected visual acuity; OCT: optical coherence tomography; logMAR: logarithm of the minimum angle of resolution.

Chronic optic nerve compression can cause axon loss, optic disc cupping, and RNFL or macular ganglion cell

thinning [1,16-19]; Portney and Roth attributed the cupping in their patient to prelaminar axon loss and glial column collapse [9]. Cupping and RNFL defects should therefore be interpreted together with visual acuity, pupillary responses, disc color, visual field distribution, the clinical course, and the severity of visual field loss relative to the structural damage. An apparently glaucomatous OCT pattern does not exclude coexisting compression.

Coexisting glaucoma cannot be excluded in our patient. The patient reported prior follow-up for a right RNFL defect, although its earlier location and any associated visual field abnormality could not be verified. The localized inferotemporal RNFL defect and the corresponding superonasal field loss observed at our assessment could represent preexisting glaucomatous damage of the NTG type, as in the patient reported by Ashok et al. [14]. It could also have been an earlier manifestation of the same compressive process, and the available data cannot distinguish these possibilities. Because the onset of each abnormality is unknown and postoperative ophthalmic examination data are unavailable, we cannot determine how much of the damage was attributable to compression. The aneurysm was therefore associated with, rather than proven to have caused, all of the findings.

Visual field defects that respect the vertical meridian are characteristic of chiasmal compression, but aneurysms near the ophthalmic artery origin or supraclinoid internal carotid artery can compress one optic nerve and produce unilateral arcuate or nasal defects that resemble glaucoma [1,9-14]. Neither the absence of bitemporal hemianopia nor a glaucomatous field pattern therefore excludes aneurysmal compression. Brain MRI with vascular imaging should be considered when the clinical course or accompanying findings raise suspicion.

This report has limitations. The ophthalmic assessment at our institution was limited to a single pretreatment visit. The clinic that had followed the patient was not the referring clinic, and its records were not available. No postoperative ophthalmic examination data were available. Consequently, neither progression nor the visual response to treatment could be assessed objectively. Color vision and critical flicker frequency were not tested, and the RAPD was not quantified. These gaps limit the characterization of optic nerve dysfunction and the separation of compressive from possible coexisting glaucomatous damage.

Conclusions

In patients with a glaucoma-like RNFL defect or visual field loss, three features should raise the possibility of a nonglaucomatous cause, including aneurysmal optic nerve compression, and lower the threshold for neuroimaging: unilateral or markedly asymmetric damage at normal IOP, a recent change in vision, and visual field loss out of proportion to the optic disc appearance and peripapillary RNFL thickness. In this patient, a new subjective deterioration over several days and a mild RAPD at normal IOP prompted neuroimaging. Imaging demonstrated compression of the right optic nerve by a supraclinoid internal carotid artery aneurysm. In retrospect, severe visual field loss with a normal-appearing disc and preserved average RNFL thickness was a further clue. The RAPD was expected from the asymmetry of the damage and did not by itself indicate its cause. The recent symptomatic change was the more informative clue.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Yasuyuki Takai, Akiko Yamagami, Kenji Inoue

Acquisition, analysis, or interpretation of data: Yasuyuki Takai

Drafting of the manuscript: Yasuyuki Takai

Critical review of the manuscript for important intellectual content: Akiko Yamagami, Kenji Inoue

Supervision: Akiko Yamagami, Kenji Inoue

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. Institutional Review Board of Inouye Eye Hospital issued approval 202606-4. The retrospective review was approved by the Institutional Review Board of Inouye Eye Hospital (approval number: 202606-4) and was conducted in accordance with the Declaration of Helsinki and relevant institutional policies. Written informed consent was obtained from the patient for publication of her clinical information and accompanying images. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All

authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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