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Delayed Presentation of Nonarteritic Anterior Ischemic Optic Neuropathy After Cataract Surgery in a Patient With Bilateral Congenital Cataracts

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Abstract

Nonarteritic anterior ischemic optic neuropathy (NAION) is the most common acute optic neuropathy in adults and has been reported after cataract surgery. Although the relative risk may increase postoperatively, the absolute risk remains low. Establishing a clinical diagnosis is difficult when the first evaluation occurs after optic disc edema has resolved. We describe a 55-year-old man with bilateral congenital cataracts and hypertension who underwent uncomplicated phacoemulsification with intraocular lens implantation in the right eye, resulting in improvement in best-corrected visual acuity from 20/80 to 20/30. Approximately one month after surgery, he noticed intermittent blurring in the operated eye, which progressed to painless loss of vision, but he did not seek care until eight months after surgery. At that time, the right eye had no light perception (NLP), a dense relative afferent pupillary defect, and diffuse optic disc pallor without edema. Optical coherence tomography (OCT) showed diffuse peripapillary retinal nerve fiber layer (RNFL) and macular ganglion cell-inner plexiform layer thinning in the right eye. The left fundus could not be visualized through the congenital cataract. Fluorescein angiography showed normal retinal arterial filling, no choroidal filling delay, and no disc leakage. Neuroimaging and serologic evaluation for arteritic, inflammatory, demyelinating, and infectious causes were unremarkable, and over 10 months of follow-up, there was no fellow-eye involvement or any features of giant cell arteritis. The findings were most consistent with NAION temporally associated with cataract surgery, occurring in an eye without disc-at-risk anatomy. New painless visual loss after cataract surgery requires prompt evaluation; a delayed presentation means the window to document acute disc edema has passed, which reduces certainty. Furthermore, profound visual loss demands rigorous exclusion of arteritic anterior ischemic optic neuropathy and other causes.

Categories: Ophthalmology
Keywords: cataract surgery, congenital cataract, delayed presentation, naion, visual loss

Introduction

Nonarteritic anterior ischemic optic neuropathy (NAION) is an acute, typically unilateral ischemic optic neuropathy resulting from hypoperfusion of the anterior optic nerve head [1,2]. The condition is characterized by sudden, painless visual loss, a corresponding visual field defect (most commonly altitudinal), and a relative afferent pupillary defect, with optic disc edema present during the acute phase [1,3,4]. Diagnosis is based on clinical findings and requires exclusion of arteritic anterior ischemic optic neuropathy as well as of compressive and inflammatory optic neuropathies [1,5]. Patients first evaluated after the acute phase, when the optic disc edema characteristic of NAION has typically resolved and optic disc pallor has developed, present a major diagnostic challenge because the defining acute findings are no longer evident [1].

Population-based studies in the United States estimate the annual incidence of NAION to be approximately 2-10 cases per 100,000 adults aged 50 years or older [1,6-8]. Established risk factors include a small, crowded optic disc (referred to as a disc-at-risk), age greater than 50 years, hyperopia, and systemic vasculopathic conditions such as hypertension, diabetes mellitus, and dyslipidemia [1,9]. Obstructive sleep apnea is recognized as a strong independent risk factor [10,11], and phosphodiesterase-5 inhibitors have also been implicated in epidemiologic studies [12]. These systemic risk factors are also present in most patients who develop NAION after cataract surgery, so their presence does not by itself argue against a postsurgical association.

Cases of NAION have been documented following intraocular surgery, most commonly after cataract extraction, referred to as post-cataract surgery NAION (psNAION) [13]. Two distinct patterns have been identified: an immediate form that occurs within hours of surgery, frequently associated with intraocular pressure elevation or systemic hypotension, and a delayed form that develops days to months postoperatively, with less clearly defined precipitating factors [1,13]. Cohort and meta-analytic studies show a modestly increased risk of NAION after cataract surgery [14,15]. In a tertiary center cohort, McCulley et al. reported 3 cases in 5,787 cataract extractions within 1 year, corresponding to a 6-month incidence of 51.8

per 100,000, which exceeds the expected background rate [16]. Nevertheless, psNAION remains uncommon in absolute terms.

This report presents a patient with bilateral congenital cataracts who developed NAION in the operated eye within weeks of phacoemulsification but underwent ophthalmic evaluation 8 months later, after the window for documenting acute optic disc edema had closed. The case illustrates how a delayed presentation compromises diagnostic certainty in NAION and underscores the need for prompt evaluation of new visual symptoms after cataract surgery, even in the absence of recognized anatomical risk factors.

Case Presentation

A 55-year-old man presented with painless loss of vision in the right eye. His medical history included hypertension, treated with lisinopril and documented as controlled, with good adherence noted in his primary care records. He had no history of hyperlipidemia or diabetes and had never smoked. The ocular history was notable for bilateral congenital cataracts. He had no history of amiodarone or phosphodiesterase-5 inhibitor use.

Eight months before this presentation, he underwent uncomplicated phacoemulsification with posterior chamber intraocular lens implantation for a visually significant cataract in the right eye. Preoperative optical biometry of the right eye showed an axial length of 24.13 mm, an anterior chamber depth of 3.84 mm, and a mean keratometry of 42.00 D. Preoperative best-corrected visual acuity was 20/80 in the right eye and 20/50 in the left. One month after surgery, the right eye's best-corrected visual acuity was 20/30; that of the left eye, which was not operated on, remained 20/50. Postoperative intraocular pressures were within normal limits, and no perioperative hypotension or intraocular pressure elevation was recorded.

Beginning approximately one month after surgery, the patient noticed intermittent blurring of vision in the right eye and, some weeks later, awoke with painless loss of vision in that eye. He did not seek ophthalmic care at that time. As a result, visual acuity and fundus appearance were not documented at symptom onset. He denied, both at symptom onset and at presentation, jaw claudication, scalp tenderness, headache, unintentional weight loss, limb weakness, diplopia, ocular pain, and trauma.

At presentation, eight months after surgery, blood pressure was within normal limits. Best-corrected visual acuity was no light perception (NLP) in the right eye and 20/50 in the left. A dense (3+) right relative afferent pupillary defect was present. Ishihara pseudoisochromatic plate testing was normal in the left eye (14/14), whereas the right eye could not be tested because of the absence of light perception. Extraocular motility was full, ocular alignment was orthophoric, and external examination showed no ptosis or proptosis. Intraocular pressures were 12 mmHg in the right eye and 14 mmHg in the left. An anterior segment examination showed a well-positioned posterior chamber intraocular lens in the right eye; the left eye was phakic with a dense, visually significant congenital cataract. There were no signs of anterior chamber inflammation in either eye.

A dilated fundus examination and ultra-widefield photography of the right eye (Figure *1A*) showed diffuse optic disc pallor without edema, a clinical cup-to-disc ratio of 0.4, attenuated retinal arterioles, and normal macula, retinal vasculature, and periphery, with no retinal whitening, cherry-red spot, hemorrhage, or visible embolus. The posterior pole of the left eye could not be visualized because of the density of the congenital cataract (Figure *1B*).

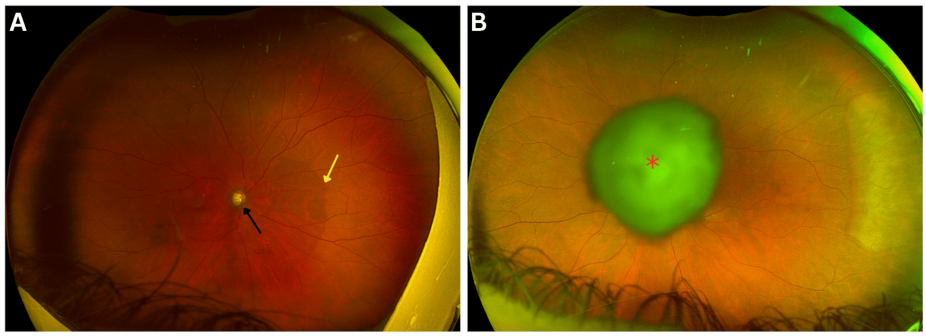
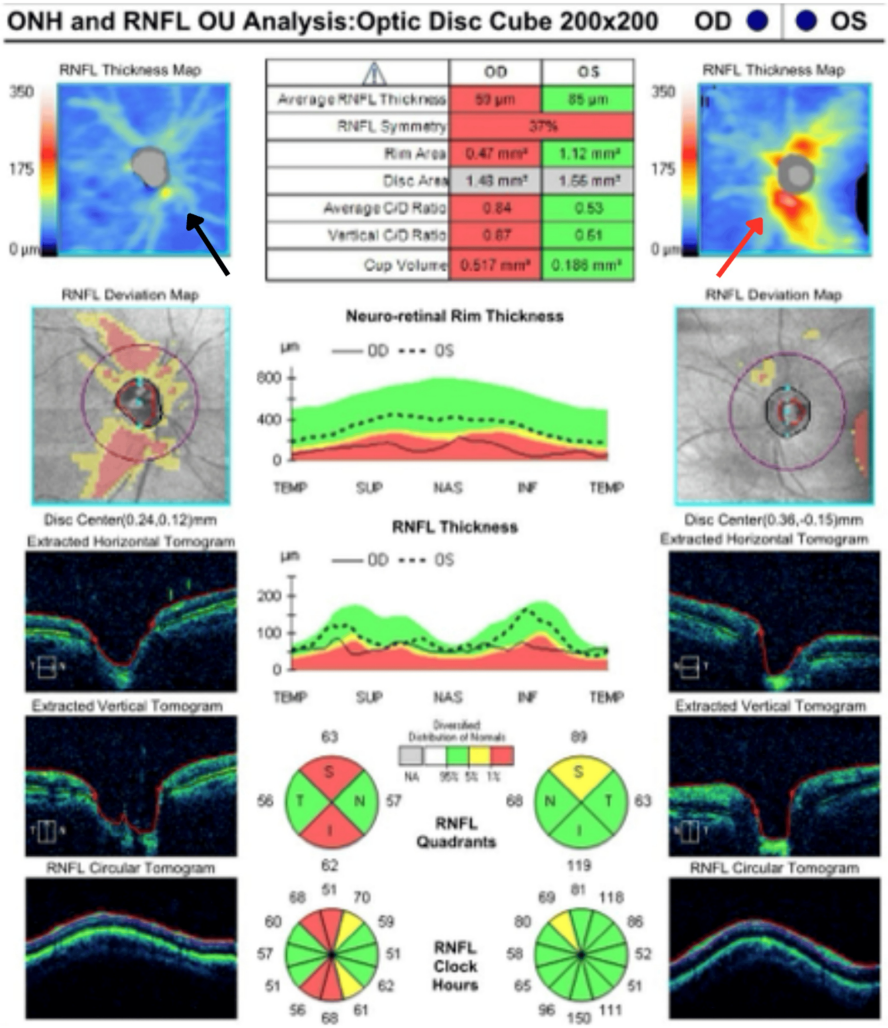


FIGURE 1: Widefield color fundus photographs (Optos) of both eyes

(A) Right eye: diffuse optic disc pallor (black arrow) without edema and attenuated retinal arterioles (yellow arrow). The macula and periphery are otherwise unremarkable, with no retinal whitening, cherry-red spot, or embolus. (B) Left eye: the posterior pole cannot be visualized because of the density of the congenital cataract (asterisk).

Spectral-domain optical coherence tomography (OCT) of the optic nerve heads (Figure *2*) showed diffuse

thinning of the peripapillary retinal nerve fiber layer (RNFL) in the right eye (average: 59 µm, superior: 63 µm, inferior: 62 µm, temporal: 56 µm, nasal: 57 µm; all below the first percentile of the normative database) compared with an average RNFL thickness of 85 µm in the left eye. The right optic nerve head had a disc area of 1.43 mm² and a rim area reduced to 0.47 mm²; the OCT-derived cup-to-disc ratio of 0.84 overestimates the clinically observed value because automated segmentation includes the atrophic rim as part of the cup. The left optic nerve head had a disc area of 1.55 mm², a rim area of 1.12 mm², and an average cup-to-disc ratio of 0.53 (vertical: 0.61), all within normal limits.



	OD	OS
Average RNFL Thickness	59 µm	85 µm
RNFL Symmetry	37%	
Rim Area	0.47 mm²	1.12 mm²
Disc Area	1.43 mm²	1.55 mm²
Average C/D Ratio	0.84	0.53
Vertical C/D Ratio	0.87	0.61
Cup Volume	0.517 mm³	0.186 mm³

FIGURE 2: Spectral-domain OCT of the optic nerve head and RNFL of both eyes

Right eye (black arrow): the average RNFL thickness is 59 µm, and the rim area is 0.47 mm². Left eye (red arrow): the average RNFL thickness is 85 µm, the rim area is 1.12 mm², and the average cup-to-disc ratio is 0.53. The OCT-derived cup-to-disc ratio of the right eye (0.84) may reflect automated misclassification of the atrophic neuroretinal rim as part of the optic cup, resulting in an overestimation compared with the clinically assessed cup-to-disc ratio of 0.4.

OCT: optical coherence tomography, RNFL: retinal nerve fiber layer

Macular OCT (Figure 3) showed ganglion cell-inner plexiform layer thinning in the right eye (average: 55 µm, minimum: 46 µm; below the first percentile in all sectors). In the left eye, the average was 33 µm; however, the signal strength score was 3/10. Formal automated perimetry of the right eye was not possible because of the absence of light perception; the left visual field showed a generalized reduction in sensitivity attributed to the congenital cataract.

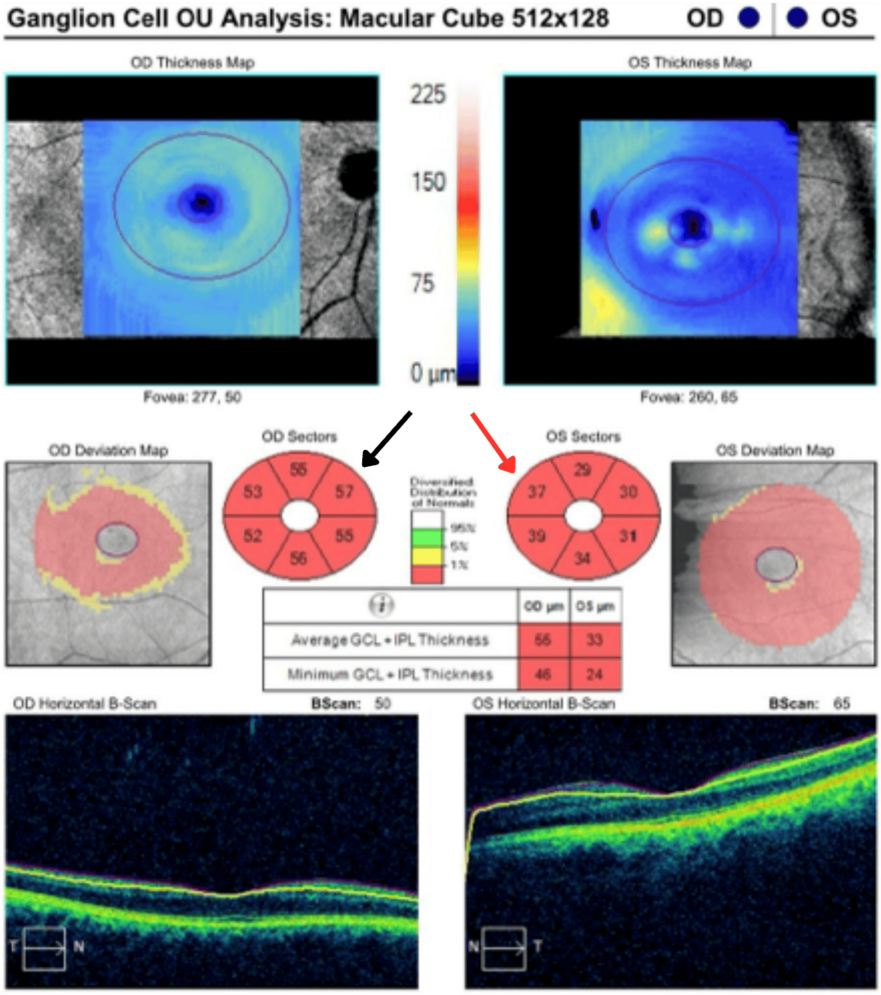


FIGURE 3: OCT macular ganglion cell-inner plexiform layer analysis of both eyes

Right eye (black arrow): average thickness is 55 µm (minimum 46 µm), falling below the first percentile in all sectors. Left eye (red arrow): average thickness of 33 µm is unreliable because of poor signal strength (score: 3/10).

OCT: optical coherence tomography

Fluorescein angiography of the right eye (Figure 4) showed normal retinal arterial filling, no delay in choroidal filling, and no optic disc leakage.



FIGURE 4: FA of the right eye

Retinal arterial filling is normal, there is no delay in choroidal filling, and the optic disc shows no leakage.

FA: fluorescein angiography

Magnetic resonance imaging (MRI) of the brain and orbits with and without contrast showed no intraorbital or retrobulbar mass, no abnormal postcontrast enhancement, and no evidence of recent infarction. A laboratory evaluation to exclude arteritic, inflammatory, demyelinating, and infectious etiologies included erythrocyte sedimentation rate, C-reactive protein, platelet count, complete blood count, antinuclear antibodies, antineutrophil cytoplasmic antibodies, IgG4, aquaporin-4 IgG, myelin oligodendrocyte glycoprotein (MOG) IgG, rapid plasma reagin with confirmatory treponemal testing, fluorescent treponemal antibody absorption, Lyme serology, and *Bartonella* serology; all were within normal limits. HbA1c and the lipid profile were within normal limits. Carotid imaging and echocardiography were not performed. A temporal artery biopsy was considered but deferred because of normal inflammatory markers and low clinical suspicion. Ambulatory polysomnography to assess for obstructive sleep apnea was recommended, but the patient declined.

At the most recent follow-up, approximately 10 months after the initial evaluation, visual acuity in the right eye remained NLP with no interval change in the optic disc. Best-corrected visual acuity in the left eye was 20/50, and the left optic nerve showed no evidence of sequential optic neuropathy. Because cataract surgery carries an increased risk of sequential NAION in the fellow eye, the congenital cataract in the left eye was intentionally left in situ after the potential visual benefit was weighed against this risk through shared decision-making with the patient.

In summary, painless visual loss beginning about one month after cataract surgery, a dense relative afferent pupillary defect, optic disc pallor, OCT evidence of RNFL and ganglion cell loss, and unremarkable neuroimaging, angiography, and serology findings were most consistent with NAION temporally associated with cataract surgery. Because the patient presented long after the acute phase, the initial optic disc edema had already resolved by the time of the evaluation and could not be documented.

Discussion

A central aspect of this case is the delayed presentation. Upon initial neuro-ophthalmic examination, optic disc edema, the documentation of which is required during the acute phase of NAION, had resolved and been replaced by pallor; furthermore, visual acuity at symptom onset was not documented. The diagnostic window for detecting disc edema in NAION is typically 4-8 weeks from symptom onset; beyond this window, optic disc pallor becomes the only fundoscopic finding [1,17]. This case illustrates a recognized but often underappreciated scenario in which a delayed presentation directly reduces diagnostic certainty.

Profound visual loss to NLP is uncommon in NAION and more typical of arteritic anterior ischemic optic

neuropathy, so giant cell arteritis required rigorous exclusion [1]. Several findings argued against arteritis: there were no symptoms of giant cell arteritis, and the erythrocyte sedimentation rate, C-reactive protein, and platelet count were within normal limits. Fluorescein angiography showed no delayed choroidal filling. Arnold et al. [18] demonstrated that choroidal hypoperfusion in the early arteriovenous phase is a hallmark of arteritic but not nonarteritic anterior ischemic optic neuropathy, and its absence supports a nonarteritic diagnosis [1,19]. Over approximately 10 months of follow-up, the patient developed neither fellow-eye involvement nor any of the systemic manifestations of giant cell arteritis; as untreated arteritis typically declares itself within this interval, this clinical stability provides further retrospective reassurance. A temporal artery biopsy was considered but deferred because the patient's inflammatory markers and platelet count were normal, a combination that yields a documented sensitivity of 97% for biopsy-proven giant cell arteritis, and clinical suspicion was low [1]. Compressive and infiltrative lesions were excluded by neuroimaging; demyelinating and autoimmune optic neuropathies were considered unlikely given negative aquaporin-4 and MOG antibodies and an unremarkable MRI; infectious causes were excluded serologically.

Two vascular occlusive diagnoses required specific attention. Chronic central retinal artery occlusion (CRAO) can, months after the event, present with disc pallor, arteriolar attenuation, and profound visual loss, all of which are indistinguishable from optic atrophy on fundoscopy. Several findings nonetheless argued against CRAO: retinal arterial filling and arteriovenous transit on angiography were normal; there was no retinal whitening, cherry-red spot, hemorrhage, or embolus; and the pattern of OCT loss, with ganglion cell-inner plexiform layer thinning in all macular sectors and a corresponding peripapillary RNFL deficit, was that of retrograde degeneration from an optic neuropathy. High-resolution macular B-scans, which in chronic CRAO show diffuse inner retinal atrophy with loss of layer distinction, were not available for review. Consequently, the distinction of NAION from CRAO rests on the angiographic and fundoscopic findings rather than on inner retinal morphology. Arteriolar attenuation is a nonspecific accompaniment of long-standing optic atrophy of any cause. Ocular ischemic syndrome was considered unlikely given the absence of choroidal filling delay, dilated retinal veins, midperipheral hemorrhages, and anterior segment ischemia, although carotid imaging was not performed. Notably, the RNFL loss in the right eye was diffuse rather than altitudinal. While NAION classically produces a sectoral pattern, diffuse loss is expected after total infarction of the nerve head in an eye with NLP and does not, by itself, discriminate between causes of end-stage optic atrophy.

The association between the cataract surgery that the patient underwent and his optic neuropathy deserves comment. The patient developed NAION in the setting of two recognized systemic risk factors: age over 50 years and hypertension. Because either factor could have contributed to the event independently of surgery, causation cannot be established. He had no history of diabetes, dyslipidemia, or tobacco exposure, and sleep apnea was not evaluated. The temporal sequence nonetheless places symptom onset about one month after a phacoemulsification, which falls within the interval of days to months described for the delayed form of post-cataract NAION [1,13]. Furthermore, the systemic risk factors present here are shared by most patients in the cohorts from which the increased postsurgical risk was estimated [14-16]. The case is therefore reported as a temporal association. The patient's report of discovering the visual loss on awakening is also characteristic of NAION [1]. Notably, the usual structural substrate for NAION was absent: neither optic disc was small or crowded on OCT, and the axial length of 24.13 mm was not considered short [1,9,20]. Congenital cataracts are typically associated with longer axial length [21]. The absence of a disc at risk makes the temporal relationship of symptom onset to surgery more striking, but it also underlines the need for the exclusion of the alternative diagnoses described above.

This case illustrates several practical points. First, new painless monocular visual loss after cataract surgery should prompt immediate ophthalmic evaluation because a delayed presentation means the window for acute-phase assessment has already passed. Second, profound visual loss demands strict exclusion of arteritic anterior ischemic optic neuropathy, as emphasized in the current guidelines [1,5]. When the examination is remote from symptom onset, other causes of optic atrophy must also be excluded. Third, optimization of modifiable vascular risk factors and evaluation for obstructive sleep apnea, the latter a strong independent risk factor for NAION [10,11], should be pursued in all patients. Fourth, no therapy has been shown to reverse established NAION. The Ischemic Optic Neuropathy Decompression Trial showed optic nerve sheath decompression to be ineffective and potentially harmful, which is why management focuses on risk factor modification and counseling to protect the fellow eye [1]. The decision to defer left eye cataract surgery in this patient was based on the increased risk of fellow eye involvement in patients with prior NAION. Lam et al. reported acute optic neuropathy after cataract extraction in 53% of fellow eyes of patients with prior spontaneous NAION compared with 19% of controls [22]. This risk was discussed with the patient and informed the shared decision to defer surgery.

This report has several limitations: its retrospective, single-patient design; the delayed presentation, which meant the window for acute-phase examination and documentation of optic disc edema had passed; and the inability to visualize the left fundus directly because of the dense congenital cataract. As a result, the left optic nerve head morphology was assessed by OCT alone, and the left macular OCT was uninterpretable. Additional limitations include the absence of temporal artery biopsy, carotid imaging, echocardiography, and high-resolution macular B-scans, as well as the eight-month interval between symptom onset and examination, which required chronic retinal arterial occlusion to be excluded indirectly rather than by acute-phase findings.

Conclusions

The development of NAION may follow cataract surgery by weeks to months and should be suspected in any patient with new painless visual loss after cataract extraction. This case, with a presentation eight months after symptom onset and NLP, demonstrates that delayed evaluation undermines diagnostic certainty, as the window to document acute optic disc edema expires over time. This clinical experience emphasizes the need for prompt assessment of new visual symptoms after cataract surgery, the rigorous exclusion of arteritic anterior ischemic optic neuropathy in cases of severe visual loss, and preventive management of the fellow eye. In this patient, NAION developed without a disc at risk and with a normal axial length, a reminder that the absence of predisposing anatomy does not exclude the diagnosis after cataract surgery.

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: María García, Miguel García

Drafting of the manuscript: María García, Miguel García

Critical review of the manuscript for important intellectual content: María García, Miguel García

Acquisition, analysis, or interpretation of data: Miguel García

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **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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