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Non-arteritic anterior ischemic optic neuropathy secondary to Posner-Schlossman syndrome in a twenty-six-year-old female

Alexandra Schulte [a], Barry Skarf [b], Pedro Monsalve [b,*]

[a] Michigan State University Clinical Center, 804 Service Rd, East Lansing, MI, 48824, USA
[b] Henry Ford Health System, Ophthalmology Department, 2799 W. Grand Boulevard, Detroit, MI, 48202, USA

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ABSTRACT

Purpose: To describe a case of non-arteritic ischemic optic neuropathy (NAION) secondary to Posner-Schlossman syndrome in a twenty-six-year-old female.

Observations: A 26-year-old female presented with painful visual loss of the left eye, elevated intraocular pressure of 38 mmHg, and trace to 1+ anterior chamber cell. Diffuse optic disc edema in the left eye and a small cup-to-disc ratio of the right optic disc were evident. Magnetic resonance imaging was unremarkable.

Conclusions and Importance: The patient was diagnosed with NAION secondary to Posner-Schlossman syndrome, an uncommon ocular entity that can significantly affect vision. Posner-Schlossman syndrome can cause a decrease in ocular perfusion pressure involving the optic nerve and can lead to ischemia, swelling, and infarction. NAION should be considered in the differential diagnosis of young patients with sudden development of optic disc swelling and increased intraocular pressure with normal magnetic resonance imaging findings.

1. Introduction

Non-arteritic anterior ischemic optic neuropathy (NAION) is a condition that leads to ischemia and subsequent infarction of the optic nerve due to a disruption in blood supply. It has been divided into anterior and posterior forms depending on which area of the optic nerve is affected, with anterior accounting for more than 90% of cases.[1] NAION has an estimated annual incidence of 2.3–10.2 per 100,000 persons in the United States, making it the most common acute optic neuropathy among individuals over the age of 50 years.[2–4] Previous studies have shown that older age, male sex, white race, retinal venous occlusive disease, age-related macular degeneration, hypercoagulable states, hypertension, hypercholesterolemia, diabetes mellitus, cardiac disease, cerebrovascular disease, obstructive sleep apnea, amiodarone and phosphodiesterase 5 inhibitor use, systemic hypotension and optic disc drusen are risk factors for developing NAION.[5–16,20] Migraine has been thought to be an additional risk factor for younger patients.[4] While the exact pathogenesis is largely controversial, NAION is presumed to result from a hypoperfusion to the short posterior ciliary arteries that supply the optic nerve head, resulting in ischemia, optic nerve head edema, focal infarction, and consequent atrophy.[4,17,21] Patients with a small cup-to-disc (C/D) ratio, also known as a "disc at risk," are at an increased risk of developing NAION because neighboring axons in a structurally crowded optic nerve disc are more likely to undergo a microvascular compression during periods of ischemia.[1,8,10–12,19,21]

Posner-Schlossman syndrome, otherwise known as glaucomatocyclitic crisis, is a disease marked by acute, unilateral, recurrent attacks of elevated intraocular pressure (IOP) accompanied by mild anterior chamber inflammation that can potentially lead to irreversible blindness.[17,18] The exact pathophysiology is unknown, however, there have been theories ranging from autoimmune to infectious.[17] Instances of IOP elevation may lead to decreased perfusion of the optic nerve head based on the principle that eye perfusion is inversely related to IOP and directly proportional to the mean arterial pressure (MAP).[22] When pressure in the eye is elevated, it compromises blood flow, putting those with a disc at risk at a higher likelihood of developing ischemia, swelling, and infarction. While a case report of NAION in a patient with acute angle closure has been described[14] and other cases of NAION in acute glaucoma secondary to Posner-Schlossman syndrome have been described,[23–27] we report a case of a 26-year-old patient with no cardiovascular risk factors and a small C/D ratio who presented to our clinic with NAION secondary to a high IOP.

* Corresponding author. Henry Ford Health System, Department of Ophthalmology, 2799 W. Grand Boulevard, Detroit, MI, 48202, USA.
E-mail address: pedrofran85@gmail.com (P. Monsalve).

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2. Case report

A 26-year-old female with a past medical history of chronic tension headaches and past ocular history of myopia and a soft contact lens wearer, reported experiencing a dark gray cloud in the left eye accompanied by a sharp left periorbital pain. She was evaluated by her primary ophthalmologist where visual acuity was right eye (OD): 20/20 and left eye (OS): 20/60; there was no improvement with pinhole. A 2+ relative afferent pupillary defect (rAPD) was noted in the left eye. IOP was measured at 12 mmHg OD and 38 mmHg OS. Confrontation visual field testing revealed an infero-nasal defect OS. On slit lamp exam (SLE), no anterior chamber cell or anterior vitreous cell was noticed. Dilated fundus exam (DFE) showed an elevated optic disc OS. The patient was advised to go to the emergency department for evaluation of presumed optic neuritis. No IOP lowering drops were prescribed by her primary ophthalmologist. The patient was admitted to the hospital for treatment with intravenous steroids and underwent a work-up for optic neuritis. Results of a brain and orbits magnetic resonance imaging with and without contrast and T1 and T2 fat saturation views did not show optic nerve enhancement, and blood work for autoimmune as well as infectious causes including syphilis, HIV, COVID-19, Quantiferon tuberculosis, Bartonella Henselae, Neuro Myelitis Optica Aquaporin 4 IgG, Myelin Oligodendrocyte Glycoprotein IgG1 FACS, Erythrocyte Sedimentation Rate, C-Reactive Protein, and Angiotensin Converting Enzyme were negative. One day after admission, the patient reported a substantial improvement in vision OS. During her second day in the hospital, she was started on intravenous prednisolone for 3 days and a tapered dose of oral steroids for 1 week. Upon discharge, the patient was referred to neuro-ophthalmology for evaluation of optic neuritis of the left eye. At the neuro-ophthalmology clinic, the patient's exam was visual acuity: 20/20 in both eyes (OU), confrontation visual fields full OU, color plates full OU, pupils reactive OU with a 2+ rAPD OS, and pressures via applanation were 14 mmHg OD and 13 mmHg OS. The patient's SLE did not show cell or flare. DFE of the right eye showed no vitreous cell and an optic disc C/D ratio of 0.1 but was otherwise unremarkable (Fig. 1A). DFE in the left eye was notable for no vitreous cell and a small, tilted disc with the entire disc margin obscured by edema. Disc edema was most prominent superiorly and disc hemorrhages were seen at 11 and 1 o'clock (Fig. 1B). The examiner was unable to determine the C/D ratio of the left optic disc. Humphrey visual field showed an unremarkable field OD (Fig. 2A) and a left paracentral scotoma OS (Fig. 2B). Optical coherence tomography (OCT) of the right optic nerve was unremarkable, with no RNFL thickening. OCT of the left optic nerve showed increased thickness of RNFL in the superior, inferior, and temporal quadrants (Fig. 3A). Ganglion cell layer in the macula appeared normal OD (Fig. 3B) and showed thinning inferiorly OS (Fig. 3C).

The patient was diagnosed with optic neuritis of the left eye and was followed every month. At the patient's 3-month follow-up there were no changes in visual acuity, confrontation visual fields, color plates, pupils, and pressure, but the left optic disc showed resolution of edema and hemorrhages (Fig. 4A). Repeat Humphrey visual field OS showed resolution of the paracentral scotoma (Fig. 4B) and OCT nerve showed substantial thinning of superior and nasal RNFL quadrants OS (Fig. 4C) with ganglion cell layer volume largely unchanged (Fig. 4D). OCT-Angiography showed a decrease in the peri-papillary capillary density OS compared to the fellow eye (Fig. 5A and B).

Four months after the initial visit the patient presented to the emergency department complaining of foggy vision in the left eye upon waking up. She stated that symptoms were similar to the previous episode but milder. Notably, pressure was measured as 41 mmHg OS via tonopen and 35 mmHg OS via applanation. Gonioscopy revealed grade IV angles OU. A 2+ rAPD was appreciated in the left eye. Slit lamp exam was remarkable for trace to 1+ anterior chamber cell OS. DFE revealed no anterior vitreous cell and a small, tilted disc with pallor superonasally OS. C/D ratio was 0.1 OU. Given the mild amount of anterior chamber inflammation, lack of corneal edema or keratic precipitates, and elevated IOP the patient was diagnosed with Posner-Schlossman syndrome and treated with pressure lowering as well as prednisone drops.

3. Discussion

3.1. NAION and optic disc edema

Optic neuritis is usually characterized by pain with extraocular movements, headaches, rAPD, and decreased visual acuity in patients under the age of 40 years old.[28] Typically only 1/3 of patients present with optic disc swelling.[28] NAION is a sight threatening disease that is rarely on the differential diagnosis for young healthy patients. However, in a young patient with atypical optic nerve swelling with disc hemorrhages, no optic nerve enhancement on magnetic resonance imaging, and visual improvement shortly after onset of symptoms and intravenous steroids, we must think about other mechanisms for such a condition.

The most common NAION finding in symptomatic high-risk patients is diffuse or segmental optic nerve swelling with peripapillary retinal hemorrhages. It is usually associated with painless vision loss and an altitudinal visual field defect. With the improvement of MRI technology, previous studies have shown that a positive post-contrast enhancement with a negative diffuse weighted imaging signal at the intraorbital segment are consistent with NAION, while positive post-contrast enhancement and diffuse weighted imaging are more consistent with

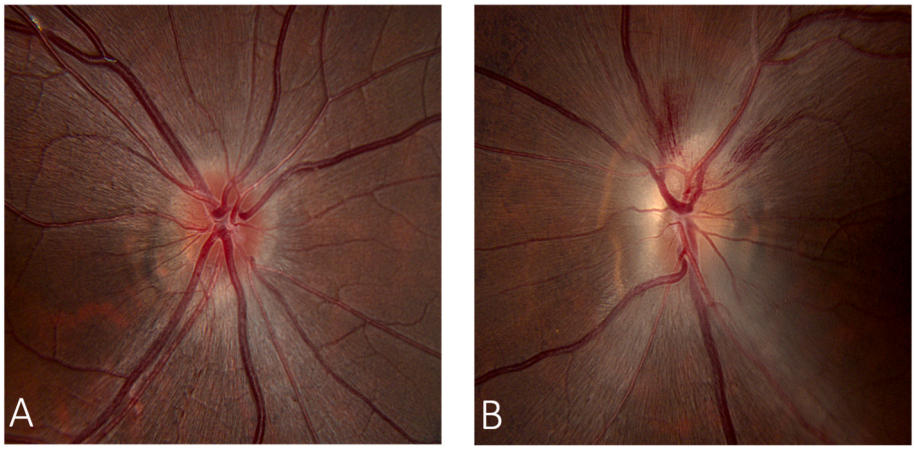

Fig. 1. A. The right optic nerve head shows a small, crowded disc with a cup-to-disc ratio of <0.1, without evidence of edema. B. The left optic nerve head shows diffuse edema most prominent superiorly, with hemorrhages at 11 and 1 o'clock.

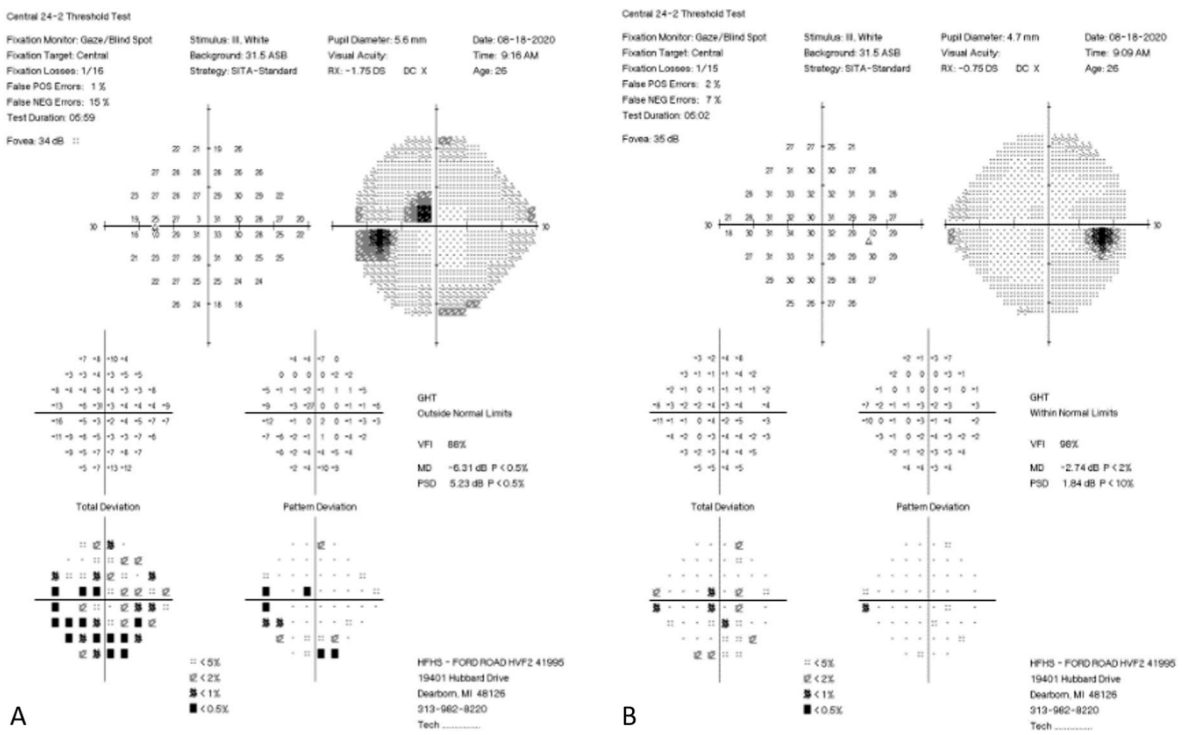

Fig. 2. 24-2 Humphrey visual field. A. Left eye: Demonstrates a paracentral scotoma. B. Right eye: Normal visual field.

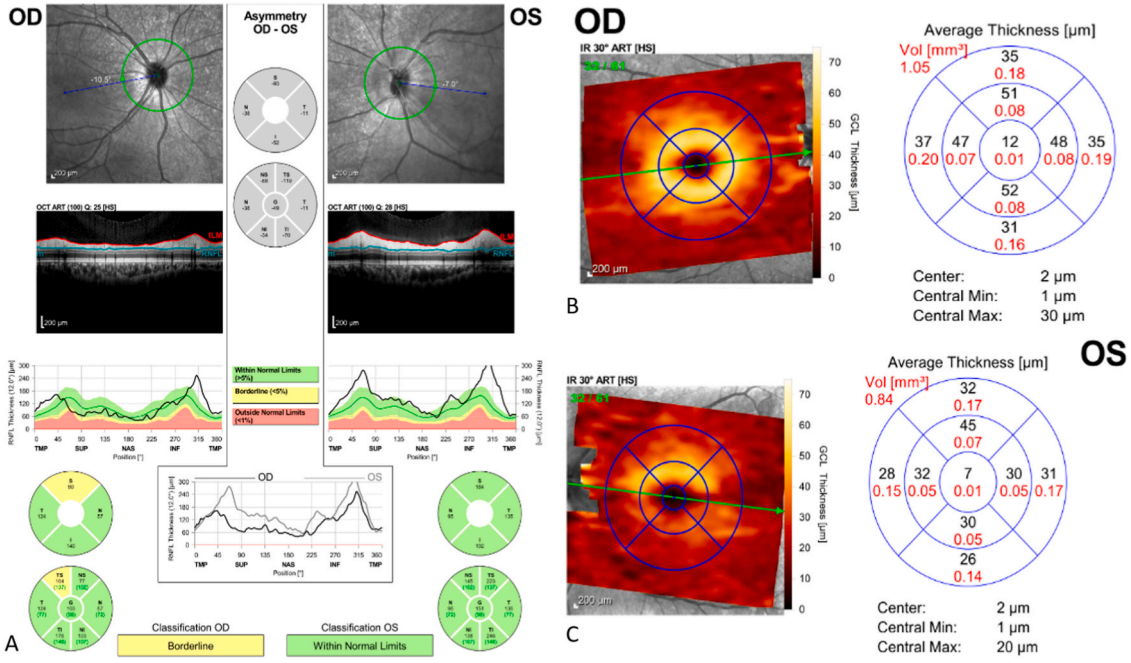

Fig. 3. A. Optic nerve OCT: Right eye: Shows no RNFL thickening. Left eye: Shows RNFL thickening superior, inferior, and temporally. B. Right eye ganglion cell layer: Shows a normal volume. C. Left eye ganglion cell layer: Shows inferior thinning. Abbreviations: OCT, Optical Coherence Tomography; RNFL, Retinal Nerve Fiber Layer.

optic neuritis.[37] Nevertheless, in most cases no optic nerve enhancement is seen on magnetic resonance imaging.[36] Therefore, unilateral optic nerve swelling without optic disc hemorrhages in younger patients is most commonly associated with optic neuritis, which typically presents with a central visual field defect accompanied by pain with eye movements, rendering NAION as a diagnosis of exclusion. Our patient presented with an atypical superior paracentral arcuate defect on her visual field that subsequently improved to a normal visual field on follow up. Previous studies have demonstrated that NAION can be present in patients under the age of 50, where chronic renal failure, diabetes mellitus, migraine and crowded discs have been reported as risk factors.[29] Nevertheless, there is no current literature describing the clinical presentation of NAION in healthy patient under the age of 35 years old.

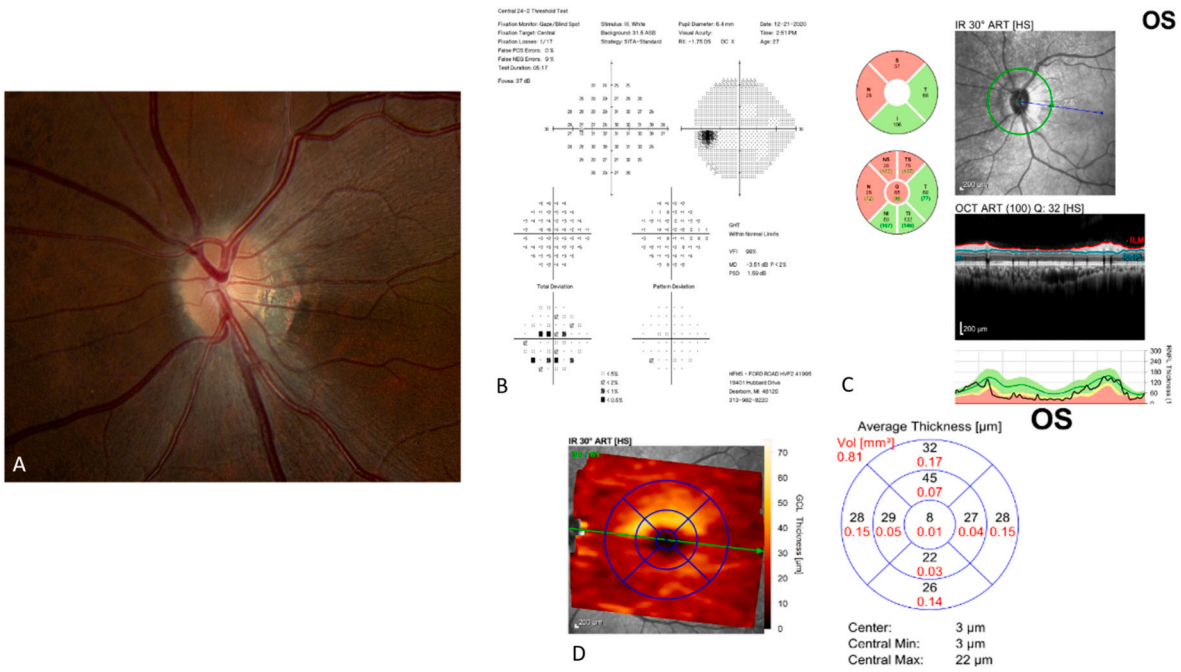

Fig. 4. A. Fundus photo shows edema and hemorrhages resolution, peripapillary atrophy with nasal, superonasal and inferotemporal RNFL thinning. B. Humphrey visual field: Shows resolution of paracentral scotoma. C. OCT nerve: Left eye shows nasal and superior RNFL quadrants thinning. D. Shows inferior ganglion cell layer thinning of the left eye.

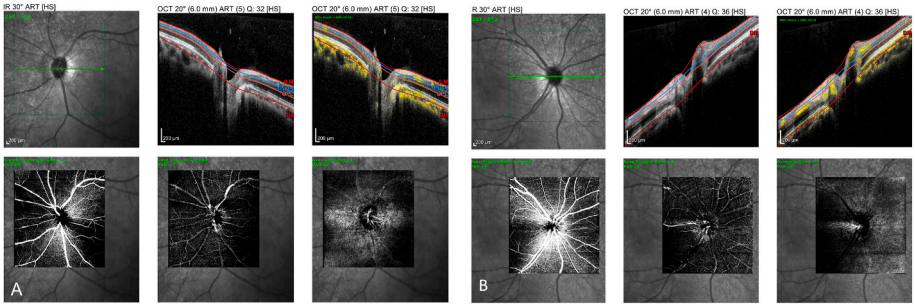

Fig. 5. A. OCT Angiography: Left eye shows decreased peri-papillary capillary density B. Right eye shows a normal peri-papillary capillary density. Abbreviations: OCT, Optical Coherence Tomography.

3.2. High IOP and NAION

An acute glaucomatocyclitic crisis may have a clinical presentation of mild eye discomfort or pain accompanied by halos and blurry vision. Patients with this condition are characterized by elevated IOP and may also have mild keratic precipitates and minimal anterior chamber cell or flare. Ocular perfusion pressure (OPP) is directly proportional to the mean central retinal artery pressure (MCRAP) and inversely proportional to the IOP. Previous studies have shown that patients with a fair C/D ratio and with multiple episodes of acute IOP increase can develop a decrease in ocular perfusion pressure, which may subsequently develop into NAION (Table 1).[26]

3.3. Ocular perfusion pressure (OPP)

OPP is defined as the pressure available to drive blood through the intraocular vasculature, with the degree of perfusion being influenced by the difference between the arterial pressure and the venous pressure.[30] This delicate balance of perfusion pressure affects the optic nerve head blood supply in that perfusion to the eye is directly proportional to the MAP and inversely proportional to the IOP.[22] Therefore, as IOP increases, perfusion to the optic nerve may subsequently decrease, leading

Table 1
Summary of previous case reports of patients with NAION associated with Posner-Schlossman syndrome including our case.

Authors	Age	Eye	Symptoms Duration	Time to NAION	Initial VA	IOP	APD	C/D	Final VA	VF defect
Kim et al.[15]	71	OS	1 week	Presentation	20/20	69	Y	0.2	20/20	Inferior altitudinal defect
Kim et al.[17]	46	OD	NR	14 years	20/20	60	Y	0.4	20/20	Superior paracentral scotoma
Irak et al.[18]	41	OD	4 days	6 months	20/50	56	Y	NR	20/25	Superior and inferior arcuate defects
Current case	26	OS	1 day	Presentation	20/60	38	Y	0.1	20/20	Superior paracentral scotoma

ADP, afferent pupillary defect; C/D, cup to disc ratio; IOP, intraocular pressure; NAION, non arteritic anterior ischemic neuropathy; NR, not registered; OD, right eye; OS, left eye; VA, visual acuity.

to optic nerve ischemia and resulting optic nerve head edema.[4,17] OPP is calculated by taking the difference between the MCRAP and IOP.[31–33] Nevertheless, measuring the MCRAP is quite problematic in a clinical setting. The MCRAP can be measured directly using ophthalmodynamometry, which is a difficult to adopt method in a clinical practice. Lovasik et al. performed a pilot study using a surrogate method to measure the MCRAP, which consisted of measuring the MAP with the arm up at eye level. The study MCRAP results were similar to those values measured with ophthalmodynamometry.[31] In addition, Kostic et al. studied a noninvasive method of measuring the OPP in 136 individuals including 30 healthy subjects, 14 glaucoma suspects, 26 open angle glaucoma, 19 Leber Hereditary Optic Neuropathy and 47 multiple sclerosis patients, which confirmed that surrogate arm up MAP correlated with the values obtained with ophthalmodynamometry.[34] The OPP is estimated first by calculating the MCRAP by using the equation MCRAP = $^{2}/_{3}$*MAP (arm up). The MAP is calculated with the equation: MAP = diastolic pressure + $^{1}/_{3}$ (systolic - diastolic pressure). We measured our patient's OPP at the time of presentation using these equations. The blood pressure in our patient with the arm at heart level was 107/87 mmHg and 93/65 mmHg with the arm up at eye level, giving us a MCRAP value of 49 mmHg ($^{2}/_{3}$*74). The OPP in our patient was derived by subtracting the MCRAP of 49 mmHg from the IOP of 35 mmHg, resulting in a value of 14 mmHg, which lies outside the normal range of 50–70 mmHg.[35] Systemic hypotension plays a crucial role in the development of NAION, [16]and a review of our patient's vitals at different times for the past 2 years provided an average blood pressure of 103/74 mmHg.

4. Conclusions

As previously described, an acute elevation in IOP can substantially decrease the ocular perfusion pressure, increasing the risk of NAION. However, according to previous studies it takes multiple acute attacks to develop NAION in patients with a fair C/D ratio. In patients with a small C/D ratio, it may only take 1–2 episodes of acute IOP elevation to develop NAION, since an elevated IOP leading to decreased OPP compromises an already crowded scleral canal, putting those patients with elevated pressures at an increased risk of compromised blood flow and frank infarction.[1,8,12] The abnormal calculated OPP value of 14 mmHg in our patient strongly suggests that ocular perfusion was impaired because of elevated IOP, leading to decreased perfusion and resulting edema. Our patient with a C/D ratio of 0.1 and elevated IOP highlights this mechanism as the probable pathophysiologic course leading to the development NAION. It is significant to note the importance of managing IOP in those patients with a disc at risk on a promptly and timely manner, as the fellow eye may later develop NAION because of its small C/D ratio. In addition, perhaps the clinician should consider starting IOP lowering therapy on patients undergoing a surgical procedure that might transiently increase the pressure in the eye. To our knowledge, this is the first case report of a healthy young patient with a diagnosis of NAION. This case outlines the importance of not excluding the diagnosis of NAION in patients younger than 50 years old with risk factors such as disc at risk and IOP spikes.

Patient consent

The patient consented to publication of the case in writing.

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Authorship

All authors attest that they meet the current ICMJE criteria for Authorship.

Declaration of competing interest

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