DOI: 10.14744/eer.2025.04834
Eur Eye Res 2026;6(1):36–43

EUROPEAN EYE RESEARCH

ORIGINAL ARTICLE

Vitreopapillary interface features in patients with non-arteritic anterior ischemic optic neuropathy

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Abstract

Purpose: The objective of this study was to evaluate the vitreopapillary interface features in patients with acute and non-acute non-arteritic anterior ischemic optic neuropathy (NAION) compared with healthy controls.

Methods: This is a retrospective cohort study and included 30 affected eyes (group 1) and 30 unaffected fellow eyes (group 2) from 30 NAION patients and 30 eyes from 30 age- and sex-matched healthy controls (group 3). Posterior vitreous detachment (PVD), peripapillary wrinkles (PPWs), and peripapillary superficial vessel protrusion (PSVP) at the vitreous-peripapillary interface were assessed from optical coherence tomography slices.

Results: The average age of the patients was 60.7±10.7 years, and the average age of the controls was 57.4±9.5 years (p=0.213). The prevalence of complete PVD at the time of acute presentation was 23.3% in group 1, 0.0% in group 2, and 16.7% in group 3 (p=0.004). At the 3rd month, the prevalence of complete PVD increased to 30% in group 1 and remained unchanged in group 2. The number of PPWs was 5.33±2.96, 3.57±1.43, and 1.37±0.76 in groups 1, 2, and 3, respectively (p<0.001) at baseline. At the 3rd month, the PPW in group 1 was significantly greater than that in group 2 (p=0.011). The number of PSVPs was 8.4±3.47 in group 1, 10.7±1.82 in group 2, and 7.43±1.28 in group 3 at baseline (p<0.001). There was no significant difference in the number of PSVPs among the groups at the end of 3-month follow-up (p=0.96).

Conclusion: PVD was present in almost one-third of eyes that had suffered NAION, and the affected eyes had PVD significantly more frequently than the unaffected eyes. The increased PPW in the affected eyes of NAION patients may be related to optic nerve head edema. NAION patients had more superficial vessel protrusion in the subacute period regardless of ischemic episode.

Keywords: Non-arteritic anterior ischemic optic neuropathy; Papillary vitreous detachment; Peripapillary superficial vessel protrusion; Peripapillary wrinkles; Vitreopapillary traction.

Non-arteritic anterior ischemic optic neuropathy (NAION) is the most common acute optic neuropathy, presenting as painless, unilateral loss of vision in the elderly population.[1,2] Presumed pathophysiology of NAION was acute hypoperfusion or circulatory insufficiency of the short posterior ciliary arteries, which is the most accepted, but no single mechanism has been fully proven.[3]

There are numerous examples of retinal damage and hemorrhages occurring during spontaneous or surgical separation of the vitreous or internal limiting membrane. Disc and peripapillary tissue also are known to be susceptible to vitreous separation.[4] Parsa and Hoyt[4] ignored the microvascular ischemic theory as a primary pathogenic mechanism. Rather, they proposed a purely

Cite this article as: Celik Dulger S, Aksu SI, Ilhan B. Vitreopapillary interface features in patients with non-arteritic anterior ischemic optic neuropathy. Eur Eye Res 2026;6(1):36–43.

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Submitted Date: 07.05.2025 **Revised Date:** 12.09.2025 **Accepted Date:** 16.09.2025 **Available Online Date:** 29.04.2026

tractional etiology causing optic neuropathy resulting from shearing forces on the optic nerve head (ONH) and peripapillary tissue during the progression of posterior vitreous detachment (PVD). In support of this theory, Modarres *et al*.[5] performed pars plana vitrectomy to release vitreopapillary attachments from the swollen ONH in eyes with NAION and reported favorable outcomes. Visual field improvement has been reported after spontaneous PVD in a patient diagnosed with NAION.[6]

One possible mechanism is that optic disc edema in NAION may be initiated by PVD, causing alterations in the vitreopapillary interface.[7] It is postulated that edema may lead to transient hypoperfusion, which further exacerbates optic disc edema. Thus, a potentially self- perpetuating cycle is initiated. Peripapillary capillary compression during PVD, further ischemia, venous stasis, and ultimately axonal damage, may be initiated by this cascade. Understanding the characteristics of patients with NAION who also develop PVD is crucial to elucidate this potential association.[8,9] Despite the proposed clinical relationship of both conditions, detailed investigations of the possible role of PVD in the context of NAION remain limited.

On the basis of the vitreous traction hypothesis proposed in the pathogenesis of NAION, this study aimed to evaluate PVD and vitreopapillary interface characteristics in acute and non-acute NAION patients compared with normal subjects using optical coherence tomography (OCT).

Materials and Methods

This was a retrospective cohort study and included data of patients with NAION who were admitted to the neuro-ophthalmology unit of a tertiary hospital in Ankara between January and September 2023. The study was approved by the local ethics commission (ethics number: AEŞH-BADEK-2024-932) and was conducted in accordance with the Declaration of Helsinki.

The diagnosis of NAION was based on the presence of several key clinical features, including sudden visual loss, color vision impairment, a relative afferent pupillary defect, a characteristic visual field defect, and associated optic disc edema. Eyes were excluded if they presented more than 3 weeks after the onset of symptoms. All potential causes of optic neuropathy, including inflammatory, hereditary, traumatic, toxic, and other factors, were excluded. The exclusion criteria for both the NAION and control groups were as follows: Retinal pathology, high myopia, glaucoma, significant media opacity affecting image quality on OCT, history of previous ocular trauma or surgery, and axial length (AL) exceeding 25.5 mm. Information was also collected regarding the subjects' medical history and smoking status.

The healthy control subjects were recruited from patients who presented to the outpatient clinic for refractive problems. The inclusion criteria were best corrected visual acuity (BCVA) ≥20/20; normal slit-lamp biomicroscopy and fundus examination; a normal visual field; a normal-appearing ONH; and no history of ocular, neurologic, or systemic disease.

All subjects (both at baseline and at 3 months for NAION patients) underwent a comprehensive ophthalmological examination. This included BCVA, intraocular pressure (IOP) measurement, and slit-lamp biomicroscopy. A dilated fundus examination was conducted with a +90 diopter lens, and color vision was assessed using the Ishihara color blindness test plates. In addition, AL and anterior chamber depth were evaluated through the Lenstar LS900 (Haag-Streit AG, Switzerland) and spherical equivalent with the Visuref 150 (Carl Zeiss Meditec AG, Jena, Germany). Visual field examinations were conducted on all NAION patients through the Humphrey Field Analyzer 3 (Carl Zeiss Meditec, Inc., USA). Incomplete and complete PVD, vitreous attachments to the ONH, peripapillary wrinkle (PPW), and peripapillary superficial vessel protrusion (PSVP) were evaluated from circular and macular OCT slices.

Optic disc and macular OCT images were obtained through the Heidelberg Engineering spectral domain OCT (SD-OCT) system (Heidelberg Engineering, Heidelberg, Germany). All OCT images were acquired by a single experienced technician. Circular peripapillary and macular OCT slices were employed to assess the presence of papillary vitreous detachment, PPW, and superficial vessel protrusion at the vitreous-peripapillary interface. The definition of PVD in this study was a complete stage 4 PVD without any presence of the premacular bursa or posterior vitreous cortex on any scans of the OCT.[10] Partial PVD was defined as a sharp and discrete hyperreflective linear image with focal attachments to the ONH, reproducible on at least one other scan. PSVP is defined as the protrusion of peripapillary superficial retinal vessels from the surface of the nerve fiber layer, as observed in circular peripapillary OCT images. It is hypothesized to be the result of vitreous traction.[11] PPWs are situated in the retinal nerve fiber layer in proximity to the disc surface or at a distance of half a disc diameter from the disc edge, manifesting as closely spaced circumferential corrugations.[12] Two blinded specialists conducted independent evaluations and recorded the OCT

parameters. A different specialist conducted a separate evaluation of the data.

Statistical Analysis

The data were analyzed using IBM Statistical Package for the Social Sciences (SPSS) (International Business Machines, SPSS) Statistics 23 (SPSS Inc., Chicago, IL, USA). The data were examined for compliance with a normal distribution through the Shapiro–Wilk and Kolmogorov–Smirnov tests. For the analysis of independent variables, the independent samples *t*-test was used to determine if the independent variables fit a normal distribution, and the Mann–Whitney *U*-test was used to determine if they did not fit a normal distribution. A Chi-square test was used for the categorical variables. For the analysis of dependent variables, the dependent samples *t*-test was used to determine if the dependent variables fit the normal distribution, and the Wilcoxon signed-rank test was used to determine if they did not fit a normal distribution. The Kruskal–Wallis test was used to compare non-normally distributed data for groups of three or more. Dunn's test was used for multiple comparisons. One-way ANOVA was employed to compare normally distributed data. Bonferroni and Duncan tests were used for multiple comparisons for groups of three or more. The findings were presented as the mean±standard deviation and median (min-max) for quantitative data and as the frequency for categorical data. Statistical significance was defined at $P<0.050$.

Results

This study examined 30 affected eyes (group 1) and 30 unaffected fellow eyes (group 2) from 30 NAION patients and 30 eyes from 30 age- and sex-matched healthy control patients (group 3). Overall, 18 female and 12 male patients with a mean age of 60.7±10.7 years and 19 female and 11 male healthy controls with a mean age of 57.4±9.5 years were studied ($p=0.213$). In the NAION group, 50% of the patients had diabetes mellitus, and 56.7% had essential hypertension. NAION was detected in the right eye in 56.7% of the patients and the left eye in 43.3% of the patients. The mean BCVA in group 1 was 1.13±0.88 (LogMAR), that in group 2 was 0.34±0.38 (LogMAR), and that in group 3 was 0.27±0.27 (LogMAR). The color vision was 3.2±4.8 in group 1, 11.33±2.26 in group 2, and 12.00±0.00 in group 3. Significant difference was found between group 1 and group 2 in terms of BCVA and color vision ($p<0.001$). The spherical equivalent was 0.26±1.62 diopters (D), 0.31±1.66 D, and 0.20±1.37 D in groups 1, 2, and 3, respectively ($p=0.964$). At the first presentation, the mean disc areas of group 2 were 1.81±0.26 mm^2. The demographic and other clinical characteristics of the groups are presented in Table 1.

Complete PVD was observed in 23.3% (7/30) of the group 1 on SD-OCT at baseline. No complete PVD was observed in group 2, and 5 of 30 eyes (16.7%) presented complete PVD in group 3. Group 1 and group 2 differed significantly in terms of complete PVD ($P=0.004$). There was no significant difference in complete PVD between group 1 and group 3, and between group 2 and group 3 ($p>0.05$). There was no significant difference in terms of partial PVD among the three groups at the time of presentation ($p>0.05$) (Fig. 1 and Table 2). At the baseline, the three groups differed significantly in terms of PPW ($p<0.001$). The mean value of PPW was 5.33±2.96 in group 1, 3.57±1.43 in group 2, and 1.37±0.76 in group 3. Superficial vessel protrusion did not differ between groups 1 and 3 ($p>0.05$). Group 2 had significantly greater PSVP than did group 1 and group 3 ($p<0.001$) (Table 3).

While the complete PVD was 23.3% of group 1 at the time of presentation, it increased to 30% at the 3rd month ($p<0.001$). The prevalence of partial PVD of group 2 did not differ significantly at the end of the 3-month follow-up. At the

Table 1. Demographic characteristics of NAION patients and controls

n (%) for categorical variables	Control (*n*=30)		NAION (*n*=30)		*p*
	n	%	*n*	%	
Sex					
Male	11	36.7	12	40.0	1.000
Female	19	63.3	18	60.0	
Systemic disease (DM)					
Yes	8	26.7	15	50.0	0.111
No	22	73.3	15	50.0	
Systemic disease (HT)					
Yes	10	33.3	17	56.7	0.119
No	20	66.7	13	43.3	
Cigarette use					
Yes	12	40.0	12	40.0	1.000
No	18	60.0	18	60.0	
Continuous variables	**Mean±S.D**		**Mean±S.D**		***p***
Age[t]	57.47±9.57 58.5 (40–71)		60.77±10.71 62 (35–79)		0.213

*$p<0.05$; *t*: Independent Sample *t*-test, categorical parameters; Chi-square test. NAION: Non-arteritic anterior ischemic optic neuropathy, SD: Standard deviation

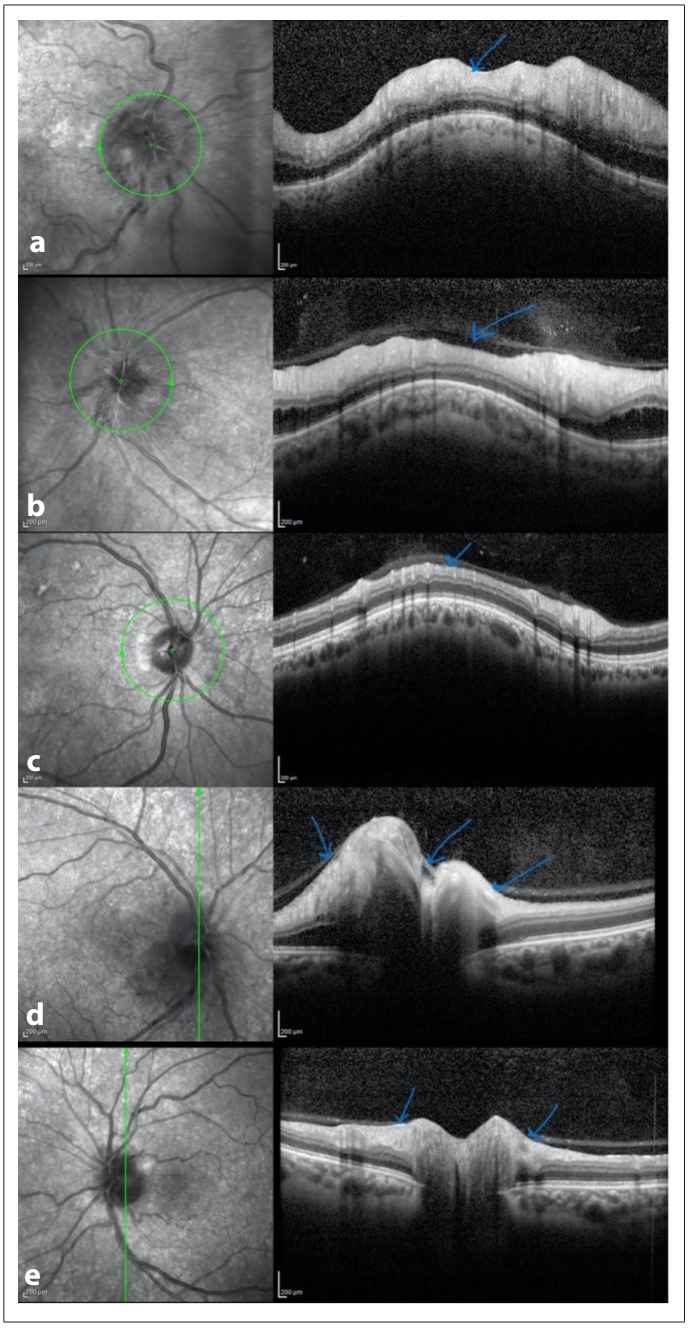

Fig. 1. Spectral domain optical coherence tomography (OCT) scan showing complete posterior vitreous detachment in a patient with acute non-arteritic anterior ischemic optic neuropathy (NAION) **(a)**, incomplete posterior vitreous detachment in a patient with acute NAION **(b)**, and no posterior vitreous detachment in a patient with acute NAION at circular **(c)**, horizontal **(d)**, vertical **(e)**, OCT slices (blue arrow).

3rd month, no significant change in the PPW was observed in group 1, whereas a significant increase in the PSVP was observed (p=0.353, p<0.001, respectively) (Fig. 2). In group 2, no significant change was observed in the complete or partial PVD at the end of the 3-month follow-up (p>0.05).

Table 2. Comparison of PVD status of affected and unaffected eyes in NAION patients at baseline and 3rd month

n (%) for categorical variables	Unaffected eye (n=30)		Affected eye (n=30)		p
	Number	%	Number	%	
PVD 0. month					
No	17a	56.7	5b	16.7	0.000**
Incomplete	13a	43.3	18a	60.0	
Complete	0a	0.0	7b	23.3	
PVD 3. month					
No	17a	56.7	4b	13.3	0.000**
Incomplete	13a	43.3	17a	56.7	
Complete	0a	0.0	9b	30.0	

*p<0.05; **p<0.01, Categorical variable: Chi-square test, a,b: There is no significant difference between groups having the same letter. NAION: Non-arteritic anterior ischemic optic neuropathy, PVD: Posterior vitreous detachment

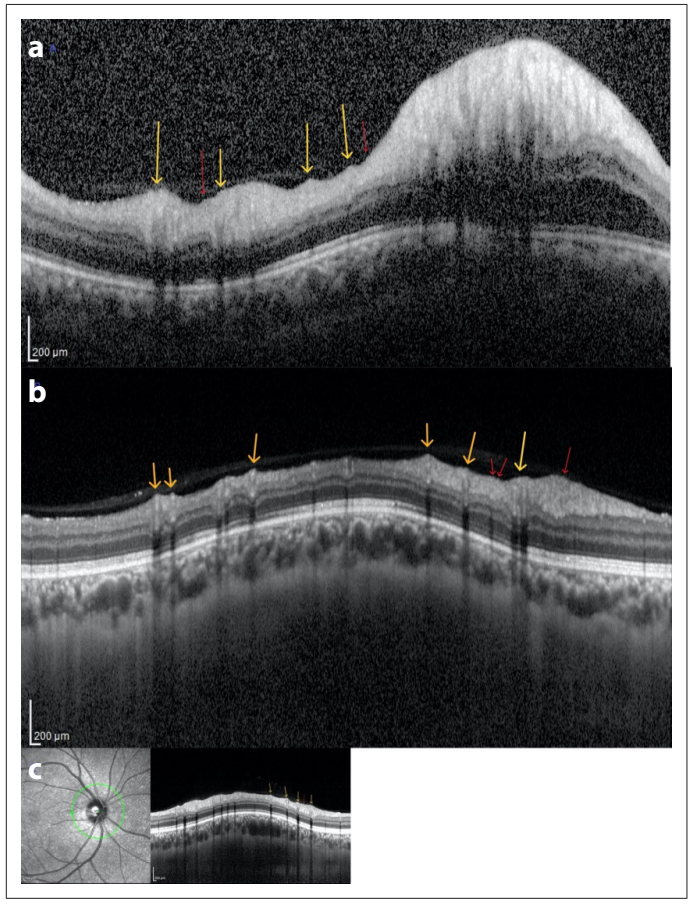

Fig. 2. Spectral domain optical coherence tomography scan showing peripapillary wrinkles (red arrow) and peripapillary superficial vessel protrusion (yellow arrow) in a patient with acute non-arteritic anterior ischemic optic neuropathy **(a)** and in the same patient at the 3rd month **(b)**, in the healthy control group **(c)**.

Table 3. Comparison of baseline ocular characteristics of the affected and unaffected eyes in the NAION group and healthy eyes

n (%) for categorical variables	Healthy eye (*n*=30)		Unaffected eye (*n*=30)		Affected eye (*n*=30)		*p*
	n	%	*n*	%	*n*	%	
Eye							
Right	30	100.0	13	43.3	17	56.7	0.000**
Left	0	0.0	17	56.7	13	43.3	
PVD							
No	8[ab]	26.7	17[b]	56.7	5[a]	16.7	0.004**
Incomplete	17[a]	56.7	13[a]	43.3	18[a]	60.0	
Complete	5[ab]	16.7	0[b]	0.0	7[a]	23.3	
Visual field							
Generalized		0	0		12		
Inferior altitudinal		0	0		11		
Superior altitudinal		0	0		4		
Quadranopic		0	0			3	
Continuous variables	**Mean±SD Med. (Min.-Max.)**		**Mean±SD Med. (Min.-Max.)**		**Mean±SD Med. (Min.-Max.)**		
Spherical equivalent[F]	0.20±1.37 −0.12 (−2–3.5)		0.31±1.66 0 (−4.25–4.5)		0.26±1.62 0 (−2.5–5)		0.964
ACD [F]	3.4±0.273.4 (3.02–4.09)		3.35±0.53 3.25 (2.5–4.6)		3.34±0.57 3.3 (2.36–4.68)		0.880
AL[F]	24.03±1.04 24.03 (22.23–26.95)[a]		23.00±0.90 22.97 (21.36–26.06)b		22.97±0.91 22.97 (21.29–26.04)b		0.000**
BCVA[H] (LogMAR)	0.27±0.27 0.2 (0–1)[a]		0.34±0.38 0.19 (0–1.4)[a]		1.13±0.88 1 (0–2.7)[b]		0.000**
Color vision[F]	12.00±0.00 12 (12–12)[a]		11.33±2.26 12 (0–12)[a]		3.2±4.8 0 (0–12)[b]		0.000**
IOP[F]	13.6±2.16 14 (10–18)		13.4±3.15 12 (9–20)		13.7±3.72 12.5 (8–21)		0.929
PPW[F]	1.37±0.76 1 (0–3)[a]		3.57±1.43 3 (2–9)[b]		5.33±2.96 5 (1–11)[c]		0.000**
PSVP[F]	7.43±1.28 8 (5–10)[a]		10.7±1.82 11 (6–15)[b]		8.4±3.47 9 (3–17)[a]		0.000**

*p<0.05; **p<0.01. Categorical variable: Chi-square test, F: One-way ANOVA test, H: Kruskal–Wallis H Test. a,b,c: There is no significant difference between groups having the same letter. SD: Standard deviation, NAION: Non-arteritic anterior ischemic optic neuropathy, PVD: Papillary vitreous detachment, ACD: Anterior chamber depth, AL: Axial length, BCVA: Best corrected visual acuity, IOP: Intraocular pressure, PPW: Peripapillary wrinkle, PSVP: Peripapillary superficial vessel protrusion, LogMAR: Logarithm of the minimum angle of resolution

Similarly, PPWs or PSVP in group 2 did not differ from the first presentation to the 3rd month (p=0.354, 0.174, respectively). At the 3rd month, a significant difference in the complete PVD persisted between group 1 and group 2 (p<0.001). The percentage of vitreous attachment to the ONH was 13.3% in group 1 and 56.7% in group 2 (p<0.001) (Table 2). The PPW in group 1 was significantly greater than that in group 2 (p=0.011). PSVP reveals no change in group 2 at the end of 3-month follow-up (p=0.96). The differences in BCVA and color vision were statistically significant in group 1 and group 2 at the 3rd month, which was similar to the findings at baseline (p=0.001) (Table 4). IOP did not differ at baseline or at 3rd month among the three groups (p>0.05).

Discussion

Our study investigated vitreopapillary interface characteristics such as PVD, PPW, and PSVP in patients

Table 4. Comparison of ocular characteristics of the affected and unaffected eyes in NAION group at baseline and 3rd month

	Month	Unaffected eye (n=30)	Affected eye (n=30)	p[1]
		Mean.±S.D	Mean.±S.D	
VA	Month 0	0,34±0,38	1,13±0,88	**0,000****
	Month 3	0,33±0,39	0,92±0,86	**0,001****
	p[2]	0,945	0,108	
Color vision	Month 0	11,33±2,26	3,20±4,80	**0,000****
	Month 3	10,67±3,28	4,03±5,16	**0,000****
	p[2]	0,324	0,205	
IOP	Month 0	13,40±3,15	13,70±3,72	0,737
	Month 3	13,87±2,96	14,00±3,35	0,871
	p[2]	0,313	0,545	
PPW	Month 0	3,57±1,43	5,33±2,96	**0,005****
	Month 3[a]	3,43±1,25	4,80±2,99	**0,011***
	p[2]	0,354	0,353	
PSVP	Month 0	10,70±1,82	8,40±3,47	**0,002****
	Month 3	10,97±1,50	10,93±3,26	0,960
	p[2]	0,174	**0,001****	

p<0,05, **p<0,01. p1: comparison of baseline and 3rd month measurements of two groups. (Independent Sample T Test, [a] Mann–Whitney U test), *p2: Comparison of baseline and 3rd month measurements in each group. (Paired Sample T Test, [a] Wilcoxon Test)), NAION: non-arteritic anterior ischemic optic neuropathy, VA: visual acuity, IOP: intraocular pressure, PPW: peripapillary wrinkle, PSVP: peripapillary superficial vessel protrusion.

with NAION. In this study, eyes with NAION had partial or complete PVD which is more common than the prevalence of PVD in the unaffected eyes. The prevalence of complete PVD increased to 30% in the affected eyes at the 3rd month visit, and remained unchanged in the fellow eyes. The prevalence of vitreous attachment was significantly greater in the unaffected fellow eyes than in the affected eyes both at baseline and 3rd month. Complete PVD showed no difference between the affected eyes and the healthy eyes.

Previous studies have suggested an association between PVD and the development of NAION.[4,13] Rabbani *et al*.[14] reported a PVD prevalence of 18.9% in patients with NAION and a significant association between PVD prevalence and advancing age. They suggested that there might not be a direct causal link between PVD and optic nerve ischemic neuropathy but rather a shared risk factor of advancing age. Others reported 30% and 25.3% of vitreous detachment in patients with NAION and a similar prevalence in the fellow eyes.[9,13] Thompson *et al*.[9] reported that complete PVD was identified in OCT sections before NAION onset in eight patients with NAION, which negates the possibility that tractional vitreous forces are involved in the pathogenesis of NAION. They proposed that if the pathogenesis of NAION was primarily mechanical, PVD should be actively developing at the time of the acute NAION. In our study, because the rates of complete PVD in the affected eyes were significantly greater than in the fellow eyes, the traction forces exerted by PVD may have facilitated the development of NAION, at least in some of the cases with small discs. Another likely speculation is that ONH edema may alter the vitreopapillary interface and trigger PVD formation. In addition, while the prevalence of complete PVD increased in the affected eyes at the end of 3 months, there was no change in the fellow eye, supporting the possibility that an acute NAION episode may contribute to the development of PVD. Shen and MacIntosh[6] reported a case developing PVD so shortly after an acute NAION episode, and her visual field worsened at the time of PVD, but improved spontaneously in a month afterward. They speculated that there is an association between two entities and suggested that vitreous traction on the ONH causes structural alterations to the axons. Over time, the release of traction on the nerve may permit restoration of normal axonal architecture and even visual recovery. If this speculation is true, triggering PVD by ONH edema secondary to acute NAION may contribute positively to visual outcomes but needs further investigation.

Previous studies have reported that vitreopapillary traction may play a role in the pathogenesis of a number of conditions such as optic nerve edema, NAION, and intra- and peripapillary hemorrhages.[4,15,16] It is a well-known condition that small and cupless discs, called disc-at-risk discs, are important in the pathogenesis of NAION. It has been shown that vitreopapillary attachments are stronger on cupless discs.[4] A recent study revealed a higher rate of focal vitreopapillary and vitreovascular attachments on crowded discs than on non-crowded discs. They suggested that crowded discs may have stronger vitreopapillary attachments, producing stronger traction forces on the vessel wall, resulting in the disruption of blood flow in NAION.[17] Similarly, Kahraman *et al*.[18] reported a strong association between vitreopapillary adhesion and crowded discs, and ONH perfusion decreased in the adhesion group. The vitreopapillary traction hypothesis proposes that traction on the ONH may cause an elongation of the nerve fibers and damage its nourishing blood vessels, resulting in disc edema due to axonal cytoskeletal damage and impaired blood flow. According to this hypothesis, ischemia still plays

an important role in the pathogenesis of NAION, but is not the primary cause of NAION.[19] Vasculopathic risk factors and characteristic sudden vision loss in NAION are indicators of ischemia. The "disc at risk" not only creates a compartment syndrome but may also contribute to strong vitreal attachments revealed in recent studies. The tractional hypothesis may be acting through the crowded disc.

A recent study suggested PPWs and PSVP as potential indicators of traction caused by papillary vitreous detachment in NAION. Kupersmith *et al*.[12] detected PPW on OCT slices in eyes with NAION and proposed that they are a common response to stress and strain resulting from swelling of the ONH and edema extending to the peripapillary retina. In our series, the PPW in the affected eyes was significantly greater than that in the fellow eyes and healthy eyes. From baseline to the 3rd month, the change in the PPW was not significant in the affected eyes or in the fellow eyes. We proposed that the increased PPW in the affected eyes of NAION patients may be related to ONH edema rather than traction due to PVD.

Li *et al*.[11] reported that 44% of eyes with acute NAION and 90% of eyes with non-acute NAION presented with PSVP. They detected a higher number of vitreous-pulled superficial vessels in the superior quadrant, consistent with more damaged visual field defects in a patient with NAION. In our study, significantly higher PSVP was observed in the unaffected fellow eyes compared to the affected and healthy eyes at the time of presentation. Three months later, PSVP in the affected eyes was similar to that in the unaffected eyes. The increased prevalence of vessel protrusion in non-acute NAION eyes observed in both our study and that of Li *et al*.[11] has been interpreted as suggestive of ONH edema in the acute period, which may potentially serve as a complicating factor in the detection of PSVP. Higher rates of PSVP in the NAION cohort compared to the healthy eyes suggest that stronger vitreopapillary adhesion in crowded discs may cause greater vitreous pulled-vessels in eyes of patients with NAION. Future studies are needed to determine whether PSVP and PPW play a role in the presumed tractional theory of NAION.

Some limitations of our study are the relatively small sample size and cross-sectional design. Another is the lack of OCT slices from patients presenting with pre-NAION status and comparison with the post-NAION appearance to interpret whether a causal relationship exists. Prospective studies with larger sample sizes and well-documented vitreopapillary interface features during acute NAION could be useful in elucidating this issue.

Conclusion

PVD was present almost one-third of eyes with NAION, and the affected eyes had PVD significantly more frequently than the unaffected eyes. The increased PPW in the affected eyes of NAION patients may be related to ONH edema rather than traction due to PVD. The presumed strong vitreopapillary attachments in crowded discs may cause greater vitreous pulled-vessels in the eyes of patients with NAION. A better understanding of the vitreopapillary status of such patients may lead to improved therapeutic and diagnostic interventions that will result in more favorable visual outcomes in this vulnerable population.

Ethics approval and consent to participate: This study was approved by the Ethical Committee of Ankara Etlik City Hospital (AEŞH-BADEK-2024-932 30/10/2024).

Peer-review: Externally peer-reviewed.

Authorship Contributions: Concept: S.C.D., S.I.A., B.I.; Design: S.C.D., S.I.A., B.I.; Supervision: S.C.D., S.I.A., B.I.; Resource: S.C.D., B.I.; Materials: S.I.A.; Data Collection and/or Processing: S.C.D., S.I.A.; Analysis and/or Interpretation: S.C.D.; Literature Search: S.C.D.; Writing: S.C.D.; Critical Reviews: S.C.D., B.I.

Conflict of Interest: None declared.

Use of AI for Writing Assistance: Not declared.

Financial Disclosure: The authors declared that this study received no financial support.

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